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DISSERTATION

ON

A CLINICAL, ENDOSCOPIC AND HISTOMORPHOLOGICAL EVALUATION OF ESOPHAGEAL LESIONS

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CERTIFICATE

This is to certify that this dissertation entitled **"A CLINICAL, ENDOSCOPIC AND HISTOMORPHOLOGICAL EVALUATION OF ESOPHAGEAL LESIONS"** is the bonafide record work done by **Dr. D. KAVITHA**, submitted as partial fulfillment for the requirements of M.D. Degree Examinations, Pathology to be held in September 2006.

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CONTENTS

INTRODUCTION

Esophageal lesions once thought to be a rare, is nowadays being one of the common disorder affecting the people throughout the world and one among the top ten malignant neoplasms affecting the people of Tamilnadu, ranking fourth in the gastrointestinal tract following stomach, colon and pancreas.

Twenty percent of esophageal tumors are benign, mostly leiomyomas, remaining 80% are malignant tumors in which about 90% are squamous cell carcinoma. The depiction of carcinoma esophagus in one of the ancient Chinese literature as “one suffers in autumn and does not live to see the coming summer”, is no more considered true, due to recent diagnostic and therapeutic advances that have improved the short term prognosis of these patients.

Esophageal cancer is a malignancy that is well known for its marked variation by geographic area, ethnic group and sex. It is one among the most dismal of visceral tumors, owing to their generally advanced stage at the time of diagnosis in developing countries.

A prospective study of patients presenting with dysphagia, loss of weight, anorexia, and regurgitation of food is undertaken to evaluate the relationship of this symptom complex to underlying esophageal lesions with special reference to Barrett's esophagus. The clinical, endoscopic findings and histopathological evaluation of esophageal biopsy specimens with routine and special stains to demonstrate the underlying nature of lesion are presented. Regarding the histopathology, previous studies on the pathologic changes of esophageal mucosa induced by Gastrointestinal Reflux Disease (GERD) has been mainly focussed and analysed.

Barrett's Esophagus (BE) is a premalignant condition in which metaplastic specialized columnar epithelium with goblet cells replaces the normal squamous esophageal epithelium, which is closely associated with GERD, in which there is a significant progression from metaplasia to low-grade dysplasia to high-grade dysplasia and subsequently to adenocarcinoma.

The prevalence of Barrett's esophagus in the general population is difficult to estimate since most cases in the asymptomatic population remain undiagnosed. The prevalence is estimated to be about 18% and may be high as 36%. Estimates of incidence of Adenocarcinoma in patients with Barrett's esophagus vary widely, but Barrett's esophagus continuous to be a major health concern in most of the western countries where numerous surveys have provided information on its epidemiology, diagnosis, and surveillance strategy.

In semiurban areas like Thanjavur even though thought to be rare, the lifestyle and nutrition factors with alcohol, a major factor, proves to be vital in the occurrence of Barrett's esophagus with subsequent adenocarcinoma. The esophagectomy specimens subjected for histopathological evaluation are also concentrated towards the histological type, morphological variants, presence or absence of changes in the upper gastric region and subsequent dysplastic changes at the adjacent mucosa. Resected studies and literature closely proves that CDC_2 / CDK_1, expression in esophageal adenocarcinoma and precursor lesions serve as a diagnostic and cancer progression marker and role of cytokeratin 7/20 in the Barrett's esophagus.

In the present study the histopathological features of Barrett's esophagus is described in detail paying particular attention to the presence of goblet cells. In addition, the recent literature regarding epidemiology, clinical features, pathogenesis and pathology of esophageal lesions is reviewed.

AIM OF THE STUDY

Esophagectomy and Endoscopic biopsy specimens were studied to find out the

1. Role of socio-economic status, epidemiology and living conditions in the occurrence of esophageal carcinomas.

2. Incidence of neoplastic and nonneoplastic esophageal lesions.

3. To evaluate site of occurrence of esophageal carcinomas.

4. To study the incidence of Barrett's esophagus and subsequently Adenocarcinoma with mucin histochemistry.

5. To evaluate the role of Immunohistochemistry in doubtful cases.

MATERIALS AND METHODS

Patients presented with symptoms and signs of esophageal lesions referred from Thanjavur Medical College Hospital and Raja Mirasudar Hospital, Thanjavur which is attached to Thanjavur Medical College during January 2000 to September 2005 were included in this study.

A thorough clinical evaluation, blood count, urine examination, Barium swallow and ultrasound abdomen (in proportion of cases) were done in each case.

A detailed history with particular attention to socio-economic status, housing conditions, nutrition, alcoholism, smoking, tobacco and betel nut chewing and history of upper Gastrointestinal complaints in other family members were also recorded.

ENDOSCOPY:

Upper gastro – intestinal endoscopy was performed with Pentax video endoscopy system 1998 E 3300 with nonspiked Pentax biopsy forceps applied with Xylocaine jelly at the tip.

Endoscopic changes were noted in the Esophagus and stomach.

Histologic study of esophageal biopsy and resected specimens: -

Two biopsy specimens were taken from the esophageal lesions which were immediately fixed in 10% buffered neutral formalin for histological evaluation.

In esophagectomy specimens the resected area was opened longitudinally from one end to another in the fresh state itself on the side opposite the tumor. The lesional area was divided into three portions as adjacent, proximal and distal to the tumor. Specimens were pinned in a corkboard with mucosal side up, allowed to float in formalin container for overnight fixation. Four longitudinal sections were taken, one including a portion of non neoplastic mucosa proximal to tumor and another distal to tumor. Two sections from the cardiac end of stomach, one including the gastro esophageal junction was taken. Proximal resected margin, distal resected margin, lymph nodes if present were also sectioned and processed. Sections taken from biopsy and resected specimens that were fixed in 10% buffered formalin were processed routinely for paraffin embedding. 3-5μm sections were cut. The sections were stained with Hematoxylin and Eosin for evaluation of

histopathologic features, (Appendix II) Alcian blue (AB), periodic acid schiff stain(PAS), (Appendix I) to demonstrate metaplasia.

Esophageal lesions were defined and classified according to the established histological criteria as per Sternberg's Diagnostic Surgical Pathology.

The presence of esophagitis, dysplastic changes, Barrett's esophagus with intestinal metaplasia, benign and malignant neoplasm were recorded in all cases of esophageal biopsies. The most prevalent appearance in each slide was matched with the standard panel formulated by renowned authors, that resembles it most closely.

The presence or absence of other histological features like fungal infections, candida, congenital abnormalties, erosions and nonspecific esophageal changes were also carefully looked for, paying particular attention to histomorphological alteration.

Histochemistry with Alcian Blue with PAS was performed to demonstrate the presence of goblet cells in Barrett's esophagus. In two doubtful cases Immunohistochemistry with markers S100, Vimentin, EMA, cytokeratin was also done.

REVIEW OF LITERATURE

Esophagus is a hollow tube connecting the mouth and pharynx with the stomach; it is composed of cervical, thoracic and abdominal segments. It possesses no significant secretory or absorptive functions and its only purpose is the transport of material from the mouth to the stomach.

Esophagus is not surrounded by a serosa; this is important clinically since once a tumor extends beyond the muscularis propria, it lies within the mediastinum.

EMBRYOLOGY:

Esophagus develops at about the 20th day of gestation when septa arising from the lateral walls of the foregut fuse, thereby separating the trachea from esophagus.

Primitive esophagus - lined by columnar epithelium, which subsequently proliferates and occludes the lumen; 7th week of gestation- esophageal lumen forms and replaced by ciliated epithelium.

5th month of gestation - stratified squamous epithelium appears in the middle third of esophagus and grows caudally and cephalad, replacing the cilated epithelium.

Esophageal mucosal glands form at 4th month of gestation.

Submucosal glands do not develop until after development of squamous epithelium.

ANATOMY

Esophagus length varies with height of the individual, ranges from 25-30cm. [5,25,29,43,51] Incisor teeth to esophagogastric junction averages 40 cm.Resting state esophagus is collapsed and measures 3 cm in lateral diameter and 2 cm in anterior posterior diameter.

UICC ESOPHAGEAL REGIONS [UICC-The International Union Against Cancer]

1. Cervical region: Extends from the cricoid cartilage to the level of thoracic inlet.

   2-4 Intrathoracic and abdominal regions.

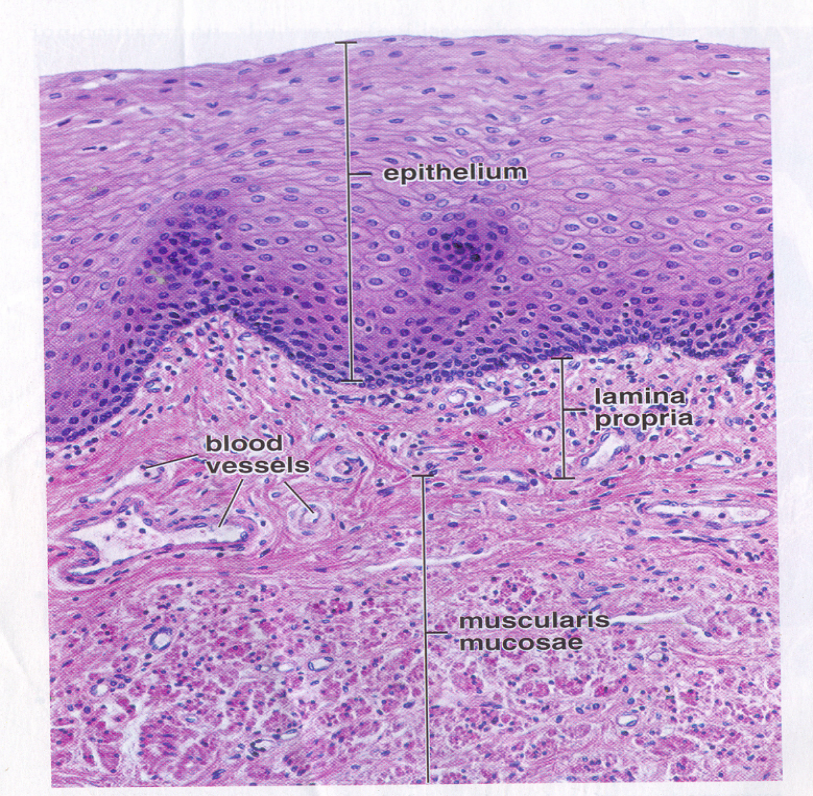
2. Upper thoracic segment: Extends from thoracic inlet to the tracheal bifurcation.

3. Mid thoracic segment: Consists of the proximal half of esophagus between the tracheal bifurcation and the esophagogastric junction.

4. Lower abdominothoracic segment: Is approximately 8 cm in length and is the distal half of the esophagus between the tracheal bifurcation and the esophagogastric junction; it includes the abdominal esophagus.

Normal Histology of Esophagus

Fig – 1



The esophagogatric junction is defined as the point at which the tubular esophagus joins the saccular stomach. The squamocolumnar junction, known as the Z-line or Ora- Serrata,[5,25,29] consists of an irregular serrated margin and does not necessarily coincide with esophagogastric junction.

Squamocolumnar junction may lie anywhere within the distal 2cm of tubular esophagus.

Histology:

The mucosa that lines the length of the esophagus has a nonkeratinized stratified squamous epithelium.

The underlying lamina propria is similar to the lamina propria throughout the alimentary tract; diffuse lymphatic tissue is scattered throughout, and lymphatic nodules are present, often in proximity to ducts of the esophageal mucous glands. The deep layer of the mucosa, the muscularis mucosae, is composed of longitudinally organized smooth muscle that begins near the level of the cricoid cartilage. It is unusually thick in the proximal portion of the esophagus and presumably functions as an aid in swallowing.

The submucosa consists of dense irregular connective tissue that contains the larger blood and lymphatic vessels, nerve fibers, and ganglion cells.

The nerve fibers and ganglion cells make up the submucosal plexus (Meissner's plexus). Glands are also present. In addition, diffuse lymphatic tissue and lymphatic nodules are present mostly in the upper and lower parts of the esophagus where submucosal glands are more prevalent.

The muscularis externa consists of two muscle layers, an inner circular layer and an outer longitudal layer. It differs from the muscularis externa found in the rest of the digestive tract in that the upper one third is striated muscle, a continuation of the muscle of the pharynx. Striated muscle and smooth muscle bundles are mixed and interwoven in the musclaris externa of the middle third of the esophagus; the muscularis externa of the distal third consists only of smooth muscle, as in the rest of the digestive tract. A nerve plexus, the myenteric plexus (Auerbach's plexus), is present between the outer and inner muscle layers. As in the submucosal plexus (Meissner's plexus), nerves and ganglion cells are present here. This plexus innervates the muscularis externa and produces peristaltic activity.

The esophagus is fixed to adjoining structures throughout most of its length; thus its outer layer is composed of adventitia. After entering the abdominal cavity, the short remainder of the tube is covered by serosa, the visceral peritoneum.

Mucosal and submucosal glands of the esophagus secrete mucus to lubricate and protect the luminal wall

Glands are present in the wall of the esophagus and are of two types. Both secrete mucus, but their locations differ.

- **Esophageal glands proper** occur in the submucosa. These glands are scattered along the length of the esophagus but are somewhat more concentrated in the upper half. They are small, compound, tubuloalveolar glands. The excretory duct is composed of stratified squamous epithelium and is usually conspicuous when present in a section, because of its dilated appearance.
- **Esophageal cardiac glands** are named for their similarity to the cardiac glands of the stomach and occur in the lamina propria of the mucosa. They are present in the terminal part of the esophagus and frequently, though not consistently, in the beginning portion of the esophagus.

BLOODSUPPLY:

Arterial blood supply derives from several primary sources. Cervical esophagus is supplied primarily by the inferior thyroid artery; the upper thoracic esophagus by the bronchial and intercostal arteries; the lower thoracic esophagus by left gastric and inferior phrenic arteries.

Numerous anastomoses connect the various arterial supplies. The venous drainages forms an extensive submucosal plexes that communicate with longitudinally oriented periesophageal veins and eventually flows into the inferior thyroid, azygous and gastric veins. In this manner, the caval and portal venous systems are connected, so that portal hypertension can result in the development of esophageal varices.

Esophagus is richly endowed with lymphatic vessels that form free anastomosing networks within submucosa and with connecting longitudinal channels in the muscularis propria.

Lymphatic flow thus tends to run longitudinally coursing cephalad in the upper 2/3rd, and caudad in the lower 3rd of esophagus, which facilitates lengthwise intramural tumor dissemination

Cervical esophagus drains into internal jugular and paratracheal nodes; the thoracic esophagus into the mediastinal and bronchial nodes; and the abdominal esophagus into a variety of subdiaphragmatic nodes. Unlike the colon, these lymphatic channels traverse the esophageal wall into the lamina propia, thus accounting for the small but definite risk of lymph node metastasis with intramucosal carcinoma.

REFLUX ESOPHAGITIS:

Casued by gastric or duodenal contents entering and injuring the esophagus

Causes: - Hiatal hernia.

Decreased lower esophageal sphincter pressure.

Endoscopy with large caliber biopsy channel and a jumbo biopsy forceps should be used

Endoscopic Findings:

Erosions, ulceration, exudates, stricture formation. Biopsy of grossly visible lesions in GERD reveals esophagitis, nonspecific injury pattern.

Pathological features:

Squamous hyperplasia[25,43] – when the length of the subepthelial papillae of the lamina propria exceeds 2/3rd s of, and the basal zone occupied more than 15% of the thickness of mucosa.

Inflammation – includes neutrophils, eosinophil and lymphocyte.

Neutrophils – differentiates normal from GERD, but nonspecific neutrophils with erosion, ulcer or associated fibrinopurulent exudates to exclude viral or candida infection.

Eosinophils – significant if more than six eosinophils are present in a biopsy section.[43]

In children – more diagnostic of GERD. In infants more than one eosinophil diagnostic of GERD

Other causes of esophageal eosinophilia :-

Eosinophilic gastroenteritis, drug reactions- Stevens Johnson Syndrome, Pill induced esophagitis.

Recent condition – Idiopathic eosinophillic esophagitis.

Lymphocytes: -

Present in large numbers in GERD No diagnostic significance because normal control subjects may have large numbers as well.

REACTIVE HYPERPLASIA AND DYSPLASIA[43]:

The most helpful histological feature are cytoarchitectural uniformity in hyperplasia versus cytoarchitectural pleomorphism in neoplastic squamous proliferations.

FEATURE	HYPERPLASIA	DYSPLASIA
Papillae	Present, fairly, regular	Absent or very irregular
Nuclear enlargement	++	+++
Nuclear pleomorphism	+	+++
Nuclear overlapping	+	+++
Nuclear hyperchromasia	+	+++
Nuclear membrane	Smooth	May be irregular

INFECTIOUS ESOPHAGITIS

Herpes esophagitis

Occurs in immunosupressed individuals.

Endoscopy:

Shallow and sharply punched out, surrounded by normal appearing mucosa.

Microscopy: Cowdry type A, dense eosionophilic intranuclear viral inclusion bodies, ground glass nuclei, nuclear molding, multinucleated giant cells, ballooning degeneration of infected cells. – in dyscohesive or multinucleated squamous cells.

Cytomegalovirus esophagitis

- Common among AIDS patients.
- Multiple well circumscribed ulcers.

CMV inclusion bodies are found in endothelial cells and fibroblasts within granulation tissue of the esophageal ulcer base and not present in the surrounding squamous epithelial cells, in sharp contrast to herpetic inclusions[43]. Presence of macrophages in a perivascular distribution, diagnostic clue to CMV esophagitis.

Candida Esophagitis

Fungal esophagitis is most commonly caused by candida albicans and candida tropicalis. Others are C. Krusei and C. Glabiata.

Because candida organisms are part of the normal flora of the GI tract, confirmation of this diagnosis requires pseudohyphae detection within the tissue more than identification of budding yeasts[43].

Endoscopy: White plaques of fibrinopurulent exudates in which pseudohyphae and budding yeast forms are demonstrated.

C.Tropicalis more virulent than C.albicans produces dysphagia and odynophagia.

Other causes of small white plaques:

1) Diffuse esophageal glycogenic acanthosis – rare manifestation of Cowden disease.
   - Distension of squamous epithelial cells with pale staining material that is PAS +ve and diastase digestible.
2) Ectopic sebaceous glands

Other Causes Of Infectious Esophagitis

- Mycobacterium tuberculosis.
- Mycobacterium Avium intra cellulare.
- Histoplasma capsulatum
- Toxoplasma gondii
- EBV
- Idiopathic esophageal ulceration in HIV.

Pill and drug induced esophagitis

NSAIDS, Antibiotics – Doxycycline, Emepromium bromide, potassium chloride, ferrous sulfate, quinidine and alendronate

Corrosive or caustic Esophageal injuries:

- Alkaline.
- Acid ingestion.

  Pathological feature are nonspecific and include tissue necrosis and subsequent inflammation.

  Long term complication – stricture formation.

Chemoradiation induced esophagitis

Initially increased apoptosis within basal zone. Bizarre epithelial and stromal cells. atypical cells with low nuclear cytoplasmic ratio.

Vascular telangiectasia is a common feature.

BARRETT'S ESOPHAGUS

Publication by N.R.Barrett's in 1950 first called attention to the columnar epithelium lined esophagus. He described the columnar lined structure as intrathoracic stomach due to congenitally short esophagus. After the columnar lined region was demonstrated to be esophagus, Barrett's proposed that the epithelium was congenital embryonic epithelium.

Available evidence indicates that Barrett's esophagus is generally acquired as a consequence of chronic gastroesophageal reflux and reflux esophagitis.

Evidences:

1) Upward migration of columnar lined mucosa over time with continuing reflux.
2) Development of Barrett's mucosa with onset of gastroesophageal reflux after esophagogastrostomy following partial esophagogastrectomy.
3) Failure to identify Barrett's esophagus in extensive autopsy studies of stillborn and neonates.

Pathogenesis:

Destruction of acid, pepsin and bile sensitive squamous lined mucosa by chronic gastroesophageal reflux followed by re-epithelization with more resistant columnar epithelium and which can undergo further metaplasia to intestinal type of differentiation.[5,25,43,51]

Cell of origin[51] – Migration of undifferentiated columnar progenitor cells from adjacent gastric and Barrett's mucosa, followed by their differentiation to columnar cells of various types.

Simultaneous migration of squamous progenitor cells from the adjacent squamous epithelium occurs, but in the abnormal milieu of ongoing reflux induced injury, the columnar progenitor cells may have selective advantage.

Totipotential cells differentiate into either columnar or squamous cells, depending upon the milieu.

Barrett's mucosa can have a wide variety of topographic appearance.

Classically, Barrett's mucosa appears as circumferential sheet resembling gastric mucosa without rugae that extends for variable distance into the esophagus about the lower esophageal sphincter.

Tongues, fingers or even islands of Barrett's mucosa involving only portions of the esophageal circumference occur frequently.

1. Some portion of Barrett's mucosa generally occurs in continuity with the true gastric mucosa.
2. Islands of squamous lined mucosa often remain within the columnar lined region, attesting to the esophageal location.

Variable components of Barrett's syndrome are peptic ulcer, stricture, reflux esophagitis and hiatal hernia.

A key aspect of the diagnosis of Barrett's esophagus is recognition that the columnar lined esophagus may be associated with none, some, or all other components of morphologic Barrett's syndrome.

Histopathologic features:

The types of cells identified in the epithelium of Barrett's mucosa include gastric type columnar mucus containing cells, which stain, with PAS and sometimes with alcian blue PH 2.5 (AB) and mucicarmine.

Columnar epithelial cells with a brush border and no mucus vacuoles, similar to small intestinal absorptive epithelial cells.

Paneth cells, with then characteristic essinophillic cytoplasmic granules, parietal and chief cells.

Neuroendocrine cells which can be argyrophil, argentaffin and enterochromaffin, which contains various hormones such as somatostatin, serotonin, substance P, enkephalin and gastrin.

Macroscopic architecture of Barrett's mucosa can include glands with deep and shallow pits, as in gastric mucosa and villous structures resembling small intestinal mucosa.

Lamina propria shows congestion, edema, acute and chronic inflammation and fibrosis.

Acute inflammation involving the epithelium can be misinterpreted as dysplasia.

Histopathologic classification of Barrett's mucosa

Category	Typical Macroscopic configuration	Prominent glandular epithelial cells	Predominant surface epithelial cells	Evidence of intestinal differentiation
Distinctive type	Villous structures and crypt like glands	Columnar mucous	Goblet interspersed among columnar mucous	Goblet cells Absorptive cells, paneth cells (variable)
Cardiac type	Glands with deep pits	Columnar mucous	Columnar mucous	Absent
Fundic type	Islands with shallow pits	Parietal, chief cells	Columnar mucous	Absent
Indeterminate type	Variable	Variable	Variable	Variable

Distinctive type Barrett's mucosa:[51]

Has villiform configuration and crypt like glands.

Glands are often continuous with muscularis mucosa and no intervening connective tissue is present.

It indicates previous ulceration of the lamina propria with epithelial regeneration directly on the smooth muscle.

Goblet cells that stain PAS, AB & Mucicarmine are both interspersed and contiguous with columnar mucous cells.

Which is identical to that of incomplete intestinal metaplasia of gastric mucosa but contrasts with that of complete intestinal metasplasia in which columnar cells are inapparent in intestinalised glands.

Distinctive type Barrett's mucosa is present in majority of adult patients with Barrett's esophagus but found less frequently in children, providing evidence for the occurrence of intestinalisation overtime.

Cardiac type Barrett's mucosa:[51]

- Resembles gastric cardiac mucosa.
- Contains deep pits which produce, villiform configuration.
- Mucus containing epithelial cells are more predominant in both surface and glandular epithelium Goblet cells are scarce.

Features of cardiac type Barrett's mucosa that differ from those of normal gastric mucosa include glandular distortion, edema and chronic inflammation.

Fundic type Barrett's mucosa:[51]

- Least common
- Shallow pits lined by mucus containing columnar cells and a heavy component of parietal and chief cells in the glands. Intestinal differentiation is absent.
- Mosaic distribution of various types of mucosa seen.

Indeterminate type Barrett's mucosa:[51]

Wide spectrum of histopathologic features.

Presence of dysplasia can lead to classifications as indeterminate type obscuring the characteristics of antecedent columnar epithelium.

Type and origin of Epithelial tumors of Esophagus:[51]

TUMOR TYPE	ORIGIN
MALIGNANT TUMORS:	
Squamous cell carcinoma	
Ordinary squamous cell carcinoma	Squamous epithelium
Verrucous carcinoma	Squamous epithelium
Basaloid carcinoma	Squamous epithelium
Spindle cell carcinoma	Squamous epithelium
ADENOCARCINOMA	
Ordinary adenocarcinoma	Columnar epithelium
Adenoacanthoma	Columnar cell with metaplasia
Mucoepidermoid carcinoma	Esophageal gland duct
Adenoid cystic carcinoma	Esophageal gland duct
Choriocarcinoma	Germ cell rest
COMPOSITE TUMORS	
Adenosquamous carcinoma	Squamous cell with metaplasia or esophageal gland duct
Carcinosarcoma	Totipotential or mixed stem cells
Small cell carcinoma and carcinoid	Foregut endocrine cell
Malignant melanoma	Melanocytes
Benign Tumors:	
Squamous cell papilloma	Squamous epithelium
Adenoma	Columnar epithelium or duct

PATHOLOGY OF SQUAMOUS CELL CARCINOMA:

The most common site of squamous cell carcinoma of the esophagus is the middle one third followed by lower and upper thirds.

The anatomic structures surrounding the esophagus are different at different levels.

Location of the tumors therefore is a major factor in determining the surgical management of esophageal SCC.

EARLY SQUAMOUS CELL CARCINOMA:[51]

Defined as a tumor that has not extended beyond the submucosa and has not metastasized.

Improved survival of patients.

Gross appearance

1. Plaque type – commonest
2. Erosive
3. Papillary
4. Occult type.

Plaque type has platform like structure, granular and white surface.

HPE:

45% - intramucosal

33% - submucosal

remainder – intraepithelial

Erosive type - depressed and eroded

Most are intra epithelial or intra mucosal.

Papillary type – elevated and eroded with polypoid or papillary contours.

Occult type – small, displays pink or congested surface, flat, difficult to recognize grossly. All are intraepithelial.

Histologically Three types:

1. Intraepithelial
2. Intramucosal
3. Submucosal

Intraepithelial carcinoma:

Malignant squamous cells involving the entire thickness of the squamous epithelium underlined by an intact basement membrane, hence squamous cell carcinoma in situ.

Intraepithelial spread of squamous cell carcinoma:

Spreading malignant cells and the apposing non neoplastic epithelium create a sharp demarcation.

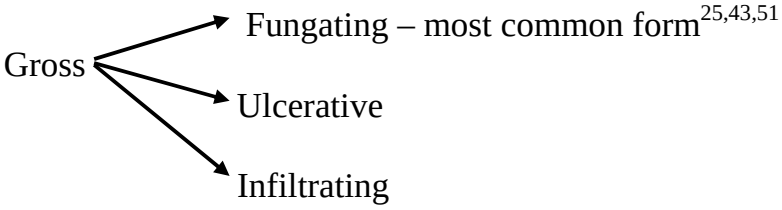
Intramucosal type of early carcinoma:

Infiltration of malignant cells in the lamina propria but not beyond. Epithelium stroma interface shows an irregular borde.

Submucosal squamous cell carcinoma:

Malignant cells have penetrated the muscularis mucosa into the submucosa but have not reached the muscularis propria. It shows frequent lymphnode metastases and lower survival rates than intraepithelial and intramucosal squamous cell carcinoma.[51]

Advanced squamous cell carcinoma:



Chinese Classification:[51]

Medullary

Fungating

Ulcerative

Scirrhous

Intraluminal (polypoid)

Microscopical Classification:

Well differentiated

Moderately differentiated

Poorly differentiated

Well differentiated SCC:

Oval or polygonal tumour cells with oval or round nuclei and prominend nucleoli. Tumour cells exhibit some degree of maturation. Keratin pearls - Keratin production in the center of tumour cell groups surrounded by flattened keratotic cells with pyknotic nuclei. Mitoses are uncommon.

Moderately differentiated SCC:

Cells are slightly smaller, more pleomorphic than well differentiated SCC. Mitoses are easily found. Focally, keratin can be observed. Tumor cells form sheets, cluster and ribbon like arrangement.

Poorly differentiated SCC:

Extremely pleomorphic cells.

Round, polygonal to spindly.

Tumors necrosis and mitoses are common.

Grades:

Grade I – Well differentiated SCC

Grade II – Moderately differentiated SCC

Grade III – Poorly differentiated SCC

Multiple squamous cell carcinomas[51]:

Second lesions tend to arise proximal to the primary SCC of the lower esophagus and distal to the upper SCC.

Criteria

a) The primary lesions shows malignant features.

b) The second carcinoma shows in situ changes.

c) At least 1.5 cm of healthy esphogeal tissue exists between these two lesions.

High prevalence of multiple esophageal SCC in alcoholics with mutant aldehyde dehydrogenase 2*2 allele.[51]

Mutated ALDH 2*2 → enzyme inactivity.

Blood level of aldehyde increases after drinking.

Multiple epithelial cells of the esophagus are aldehyde preconditioned, which may be activated with exposure to known carcinogens. (smoking etc)

→ Irreversible malignant change.

P53 alteration - key event in multifocal esophageal carcinogenesis.

Variants of squamous cell carcinoma

Verrucous carcinoma

Rare, distinct type

Gross: exophytic, cauliflower like or papillary and white.

Microscopy: superficial part of the tumor shows characteristic papillary projections, but the deeper part is composed of acanthotic squamous epithelium, compressing or infiltrating the underlying tissue.

Malignant cells are characteristically found in the basal layers.

Basaloid carcinoma:

Characterized microscopically by solid sheets or island of tumor cells surrounded by basal lamina, closely resembling features of basal cell carcinoma of the skin, or by gland like structures resembling, those of salivary gland.

The tumor cells are small, have relatively scanty cytoplasm, similar to the basal cells of squamous epithelium.

Peripheral palisading is prominent and characteristic of basaloid carcinoma with frequent mitoses and focal necrosis.

Spindle cell carcinoma and carcinosarcomas

Gross: polypoid

Tumors may reach a diameter of 15cms. No sharp demarcation between malignant spindle cell and squamous cell carcinoma components.

Osseous, cartilage and muscular tissue may be seen.

Results of light and electron microscopic and immuno histochemical studies demonstrate that these tumors are heterogeneous but can be categorised into 2 groups[51].

Carcinosarcoma: – epithelial nature of SCC and mesechymal nature of malignant spindle cell components are proven

Sarcomatoid carcinoma: -

Tumour in which both SCC and malignant spindle cells show epithelial markers.

Composite carcinomas:

Synonym- chimeric tumor. Some esophageal cancer show two or more cellular components.

Different components either intermingle or demonstrate transition from one to another.

(e.g.) Adenosquamous carcinoma

SCC with small cell carcinoma

SCC with carcinoid

Cell of origin of Adenocarcinoma[51]

Columnar epithelium of esophagus.

Congenital remnants and gastric heterotopia:

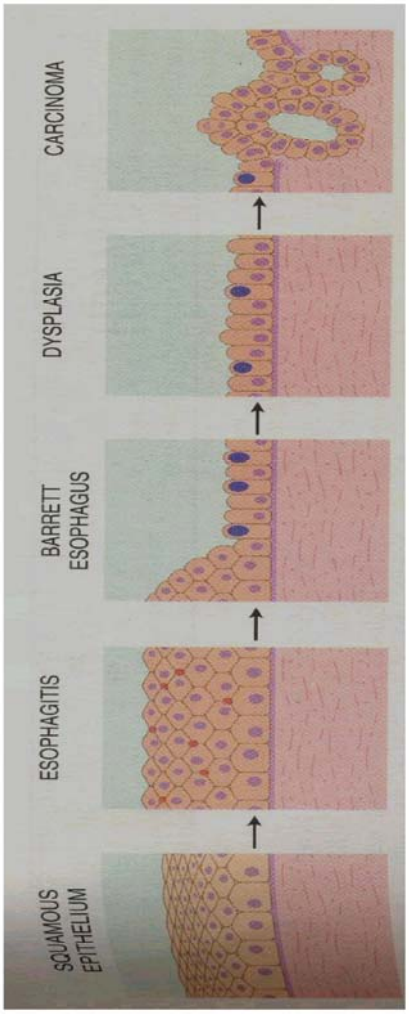
Incidence 11.8% in children and 4 to 10% in adults. These patches consists of fundic type of gastric glands with chief and parietal cells and measures 0.2 to 0.3 cm.

Barrett's epithelium:

Is pink and velvety, resembling gastric mucosa in contrast to the light gray normal squamous mucosa.

Fig – 2

Transition from barrett's esophagus to adenocarcinoma



Congenital or ectopic gastric epithelium can be readily differentiated from the Barrett's epithelium by well formed fundic glands whereas Barrett's epithelium shows intestinal metaplasia.

Submucosal esophagal glands:

Submucosal glands with ducts penetrating through the squamous epithelium into the lumen of esophagus.

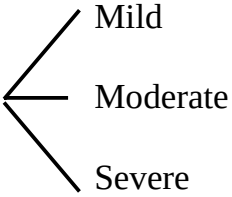
Uncommon cell types of Esophageal epithelium:

Normal esophageal squamous epithelium has scattered endocrine and melanocytes.

Endocrine cells are argyrophilic-giving rise to carcinoid and small cell carcinoma.

Melanocytes give rise to rare melanomas.

Dyplasia of Barrett's epithelium:



Low grade mild dysplasia shows decreased mucous secretion, crowding of slender columnar cells with pseudostratified nuclei and occasional nuclear pleomorphism, mitosis absent.

High grade dysplasia shows moderate pleomorphism, plump cells marked reduction of mucus secretion and frequent mitosis. Glands may show budding, branching, crowding and intraluminal folding.

ADENOMA:

Consists of dysplastic epithelium, localized growth, sharp demarcation with surrounding tissue.

ADENOCARCINOMA:

Flat, some may be polypoid and larger 4.5cm.

Most advanced tumors all flat and ulcerated [25,43,51].

1/3rd are polypoid or fungating.

Diffuse infiltrative lesions in the form of linitis plastica and gross papillary lesions are rare.

Microscopic features:

Majority are well or moderately differentiated tubular adenocarcinoma.

Microscopic classification

Well differentiated.

Moderately differentiated.

Poorly differentiated

Mucinous.

Signet ring cell type.

Lauren's classification used for gastric carcinoma can be used for adenocarcinoma of esophagus too.

Adenocarcinoma at the esophagogastric junction:

Majority are adenocarcinomas

2/3rd of adenocarcinomas arise from stomach.

1/3rd from esophagus.

Basis for differentiation is location of the center of the tumor and presence of specific structures of the respective organs[25, 43,51].

Esophagus – specialized, incompletely intestinalised epithelium gives a clue.

Adenoacanthoma and adenosquamous carcinoma

Adenoacanthoma is adenocarcinoma with focal squamous metaplasia, rare

Adenosquamous carcinoma is basically a squamous cell carcinoma with occasional mucus secreting glandular components.

Same outcome as that for tumors without the minor metaplastic components.

Mucoepidermoid carcinoma:

Arises from esophagal gland ducts. Composed of both squamous and glandular cells.

50% occur in the middle one third esophagus. Aggressive, extensive invasion and lymph node metastasis are common.

Adenoid cystic carcinoma:

Origin from the intercalated duct of submucosal esophageal glands.

Mostly in middle 3rd of esophagus in the submucosa[51].

Histologically, lesions are composed of well defined islands of tumor cells with a cribriform pattern and many cystic spaces.

Carcinoid:

Submucosal tumors, arise mostly in the distal esophagus, composed of uniform polygonal or round cells in sheets and trabeculae, with salt and pepper type chromatin containing nuclei.

Small cell carcinoma:

Arises in the middle and distal segment, second to the lung, esophagus is the most common site for small cell carcinoma[25,51].

Gross:

Large, fungating or ulcerated, polypoid form is rare.

Histologically, tumor is composed of small cells with hyperchromatic nuclei and scanty cytoplasm.

Poor prognosis.

Malignant melanoma:

Rare, about 10% of esophageal malignant tumors, frequently in lower esophagus.

Tumor is characterized by marked cellularity, frequent mitosis, presence of intra cellular as well as extracellular melanin pigments.

Junctional changes with melanoma cells at the base of the covering squamous epithelium are common.

Choriocarcinoma:

Large ulcerated tumors mainly lower segment, rare tumors composed of cyto trophoblast and syncytiotrophoblasts.

Paget's disease:

Large clear pagetoid cells found in the squamous epithelium and glandular ducts without stromal invasion.

SECONDARY AND METASTATIC TUMORS:

3% of all carcinoma show metastases to esophagus.

Direct spread: From lung or mediastinal lymph nodes.Carcinoma of the upper stomach and hypopharynx may extend into the contiguous esophagus or metastasized to esophageal wall by lymphatics.Metastatic tumors are submucosal, small and asymptomatic.Exception is metastic melanoma, forms large pigmented polypoid mass[51].

BENIGN TUMORS AND TUMOR LIKE LESIONS

Benign tumor are mostly mesenchymal benign epithelial tumors [25, 51], squamous cell papilloma and adenoma are rare.

Squamous papilloma

Sessile lesions. Its papillae are composed of a central core of connective tissue covered with hyperplastic squamous cells in an orderly arrangement and without dysplastic changes. Distal esophagus is the usual site.

Cysts:

Retention cysts formed by dilated ducts of esophageal glands are common. Submucosal, more in lower segment. Duplication cysts of developmental origin are primarily extramural and connect with the esophagus only partially if at all. Cysts may be lined by esophageal, bronchial or gastric epithelium.

Leiomyoma:

Although it is the most common benign tumor of the esophagus, leiomyoma seldom poses a clinical problem. In general these tumors appear as circumscribed mural masses, usually solitary, and 2 to 5 cm in diameter, that bulge into the lumen and may even form pedunculated polyps. Minute “seeding” leiomyomas of 1- to 2-mm diameter can be commonly identified in the vicinity of the gastroesophageal junction.

The morphologic features mirror those of other classic leiomyomas: interlacing fascicles of bland spindle cells with variables fibrosis produce the traditional circumscribed, white-gray whorled appearance on gross examination. Distinction from the very rare esophageal leiomyosarcoma and esophageal gastrointestinal stromal tumor is not difficult based upon morphologic and immunophenotypic findings.

Mesenchymal polyps:

A polypoid configuration is the most memorable feature of several benign esophageal lesions of unsettled pathogenesis. Fibrovascular polyps are characterized by a core of mature fibrous tissue, occasionally myxoid, with scattered thin walled vessels and a variable admixture of adipose tissue. The overlying mucosa is generally intact, but may be secondarily eroded.

The histologic variety explains this polyp's numerous synonyms, which include fibroma, fibrolipoma, fibromyxoma, and lipoma. Typically these polyps arsie in the upper esophagus from the cricopharynegeal region, and they present as elongated pedunculated masses that may range up to 20 cm in length.

Inflammatory fibroid polyps similar to those found else-where in the gastrointestinal tract have also been described in the esophagus. They comprise a submucosal proliferation of reactive fibroblasts and small vessels bestrewn with inflammatory fibroid polyps are located in the mid or lower esophagus and harbor a more diffuse inflammatory component, although the distinction between the two without great clinical importance.

Inflammatory esophagogastric polyps are composed of granulation tissue and edematous lamina propria with inflammation. They occur near the gastroesophageal junction and probably result from reflux –associated ulceration and repair.

Granular Cell Tumor:

The esophagus is one of the favored gastrointestinal sites for granular cell tumors[25]. Most are solitary, asymptomatic lesions that arise in the distal esophagus. However, in some cases, multiple lesions arise in the same patient, either limited to the esophagus or distributed in multiple gastrointestinal tract sites. Grossly, they appear as intramural nodules, poorly circumscribed, and generally less than 2 cm in diameter.

They resemble granular cell tumors from other sites and are composed of sheets of uniform cells with small nuclei and abundant granular PAS – positive cytoplasm that exhibits S100 protein immunoreactivity. The overlying squamous epithelium can undergo pseudoepitheliomatous hyperplasia that may be so profound as to be mistaken for squamous cell carcinoma. Most esophageal granular cell tumors are stable or slowly growing benign lesions, but rare examples with malignant behavior have been reported.

Miscellaneous:

Case reports describe all types of benign soft tissue tumours occurring in the esophagus: hemangiomas, lymphangiomas, glomus tumors, osteochondromas, and neurofibromas. Hamartomas fashioned from a mixture of fibrous, muscular, cartilaginous, and glandular tissues are found in infants.

Endoscopic picture[29]

Esophago gastric junction mucosa: also known as dentate or Z line.Columnar line segment is orange red to red, and is distinctly different from the pinkish, gray colour of normal esophageal squamous mucosa. The dentate line is sometimes regular, giving the appearance of sharp ring like demarcation. (Fig 3)

Fig 3 Endoscopic appearance of Gastroesophageal junction –red columnar mucosa with pink squamous mucosa –dentate or Z line.

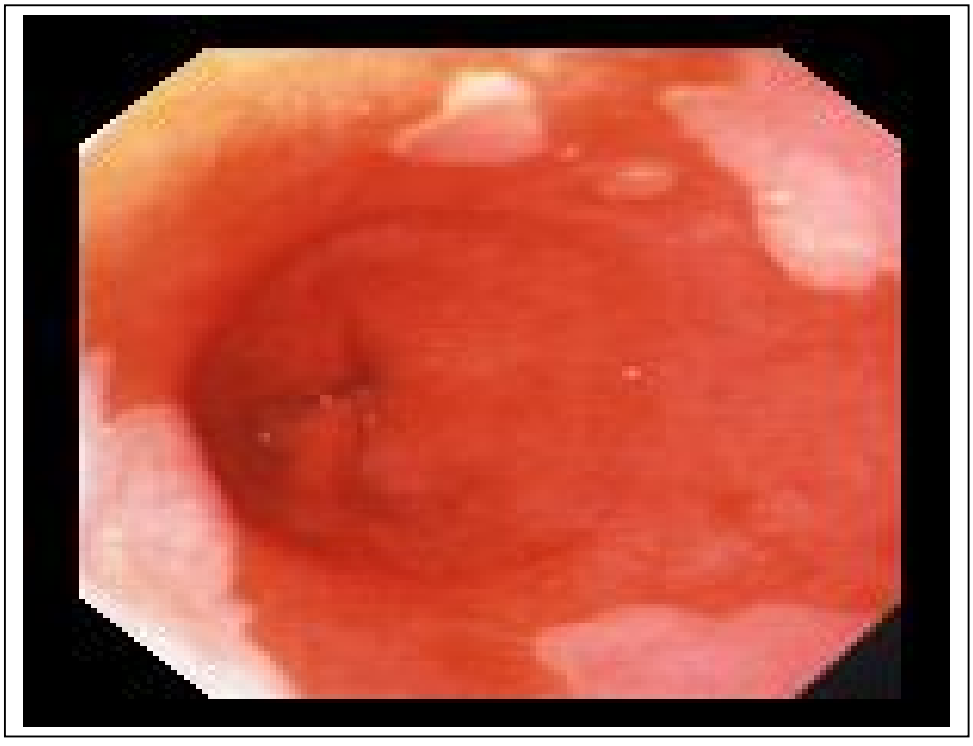


Fig 4 Endoscopic appearance of Barrett's mucosa –irregular tongues of red glandular mucosa extending up into the pink squamous mucosa.

Fig 5 Endoscopic appearance of Carcinoma esophagus-irregular obstructive growth with menisci sign.

Barrett's esophagus

The squamo columnar junction is irregular. Tongues of red columnar mucosa extends into pink squamous mucosa. (Fig 4) The extend is recorded in centimeters from the columnar junction to the point at which the lumen changes from a tubular shape into a cavity.

Carcinoma of the esophagus

Irregular, relatively firm, ulcerated surface. Some times polypoid, obstructive with menisci sign[29]. (Fig 5)

CHROMOENDOSCOPY

Chromoendoscopy[42]

Chromoendoscopy uses chemical staining agents applied to the gastrointestinal (GI) mucosa to identify specific subtypes of epithelia or to highlight surface characteristics of the epithelium. Chromoendoscopy has been used in several regions of the GI tract, including the esophagus, stomach, duodenum, and colon, to aid the characterization of multiple disease states.

Two types of tissue staining are used in the esophagus. Vital, or absorptive stains, such as Lugol's solution, methylene blue, and toluidine blue are all actively taken up by esophageal mucosa, Contrast stains are not absorbed but highlight the surface of the mucosa, allowing for the identification of minute lesions and subtle patterns. Contrast stains currently used in the esophagus include indigo carmine and dilute acetic acid solution.

Lugol's solution

Lugol's solution is an inexpensive, widely available solution comprising a mixture of iodine and potassium iodide. This vital stain is absorbed by glycogen-containing, nonkeratinized, squamous epithelium, the normal tissue type in the esophagus. Lugol's-stained tissue characteristically turns green-brown. The intensity is partly dependent on the amount of glycogen present within the epithelium. Inflammatory or dysplastic squamous epithelium, squamous cell carcinoma, and columnar epithelium do not stain with Lugol's solution. This stain is used as a 1% or 2% solution in a volume of 20 to 50 ml sprayed through endoscopic catheters.

Toluidine blue

Toluidine blue is a basic metachromatic vital stain that has been used in the evaluation of oral and female genital tract lesions and squamous cell carcinoma of the esophagus. This stain is also useful in the endoscopic detection of BE, IM was detected with 98% sensitivity and 80% specificity.

Methylene blue

Metaplastic epithelum, including IM of the stomach and esophagus, also absorbs methylene blue.

Methylene blue is not absorbed by normal squamous or gastric epithelium.

Before applying the stain, surface mucous must be removed to expose as much surface area as possible for staining.

ENDOSCOPIC BRUSH CYTOLOGY

Cytology in Barrett's esophagus[42]

Limitations of current endoscopic biopsy surveillance programs

Current guidelines suggest obtaining systematic four-quadrant biopsy specimens at 2-cm intervals along the entire length of the Barrett's segment once inflammation related to gastroesophageal reflux disease is controlled with antisecretory therapy.

Potential advantages of endoscopic brush cytology

The sensitivity of combined endoscopic biopsy and cytology is 92% to 100% for the diagnosis of gastroesophageal malignancy, which is better than biopsy or cytology alone.

Technique and processing of brush cytology

Endoscopically directed brush cytology samples should be obtained before biopsy. Cytologic samples are obtained with a cytology brush from all four quadrants along the entire length of the Barrett's segment and from any endoscopically noted abnormalities, such as ulcers, erosions, nodules, or plaques.

Cytologic specimens may be prepared as either conventional smears or with liquid-based techniques such as ThinPrep (Cytyc, Marlborough, MA). The ThinPrep process involves specimen collection directly into a methanol-based preservative solution.

This solution is centrifuged, and after discarding the supernatant, the cell pellet is resuspended and a sample transferred to a second methanol-based preservative. An automated device, the ThinPrep Processor subsequently prepares cytology slides with a thin layer of evenly dispersed cells.

Cytologic criteria in Barrett's esophagus

Cytologic specimens may be categorized as satisfactory or unsatisfactory by the presence or absence of well-visualized cellular elements. Satisfactory specimens can then be further classified into one of the three categories. Specialized columnar epithelium present, diagnostic of BE; columnar epithelium present, consistent with BE; or no evidence of Barrett's epithelium. Specialized columnar epithelium may be defined as the presence of goblet cells, identified by large well-delineated cytoplasmic vacuoles. The cellularity of specimens containing columnar cells may be subjectively evaluated as mildly, moderately, or markedly cellular.

Specimens with columnar epithelium may be classified into one of four categories: negative of dysplasia, indefinite for dysplasia, High Grade Dysplasia (HGD) and adenocarcinoma.

Barrett's epithelium negative for dysplasia typically occurs in large cohesive cellular sheets, the edges of which are sharply defined and smooth. Cellular polarity is maintained with nuclei uniformly distributed within the groups, creating a honeycomb appearance. The benign nuclei, containing finely granular chromatin, have an oval configuration with smooth external nuclear contours and vary little in size. When goblet cell differentiation is present, it usually is obvious as large well-delineated cytoplasmic vacuoles.

Nuclei are variably enlarged with aberrations of the nuclear membrane, such a thickening, notches, and irregular contours. Nucleoli are more prominent and the nuclear-cytoplasmic ratio is enlarged compared with non-dysplastic cells. Cell groups are smaller and more heterogeneous, frequently forming three-dimensional aggregates. A major difference between cytologic samples negative for dysplasia and dysplasia-adenocarcinoma specimens is the arrangement of the constituent cells.

Cells are variable in size and shape and there is a strikingly irregular and haphazard distribution of cells within the aggregates with irregular crowding and nuclear overlap.

Another prominent feature of dysplasia-adenocarcinoma is loss of intercellular cohesiveness manifested as frayed edges to the cellular groupings, with cells at the margins of aggregates appearing to fall away. Large numbers of atypical aggregates and single atypical cells and giant nuclei are considered more characteristic of adenocarcinoma than dysplasia.

The term “indefinite of dysplasia” is used for preparations with atypical cellular features that are not sufficiently developed to permit an unequivocal interpretation of HGD or adenocarcinoma or frank adenocarcinoma.

Efficacy data

Despite its successful use in other cancer programs, surprisingly little information is available on the usefulness of cytology in the diagnosis and surveillance of BE. The potential for endoscopic brush cytology was first described by Belladonna et al in a 1974 cases report of a patient with an esophageal stricture on barium radiography that could not be confirmed at endoscopy. Biopsy specimens obtained from an area of inflammation revealed columnar epithelium but no neoplasia. Endoscopic brushings and washings both demonstrated adenocarcinoma, however. Esophageal resection revealed multi-focal adenocarcinoma.

Compared with histologic analysis, the sensitivity of cytology for detecting specialized columnar epithelium was 82% and the specificity was 100%. Furthermore, Barrett's epithelium of the specialized type was detected only by cytology and not by histology in 11 patients.

Nonendoscopic techniques[42]

Given the high cost of endoscopic surveillance and the potential benefits and lower cost of cytology, blind, nonendoscopically directed balloon cytologic sampling has been suggested as a potential cost-effective alternative for cancer surveillance in patients with BE.

OBSERVATION AND RESULTS

This study covered a total of 323 cases in which 277 were endoscopic biopsies and 46 were esophagectomy specimens.

In 277 endoscopic biopsies 193 were males (69.68%) with age ranging from 20 to 80 yrs (mean age 50 yrs), 84 were females (30.33%) with age ranging from 20 to 80 yrs (mean age 50 yrs).

The clinical presentation, endoscopic picture, and histopathological features, are listed in the master chart.

Table No1 shows the total number of esophageal biopsy specimens referred for histopathological evaluation during the period January 2000-September 2005. The average incidence is 9.75%.

TABLE I

S. No	Period	Total no of Biopsies	Esophageal Biopsies	Percentage
1	Jan2000-Dec2000	410	49	11.95%
2	Jan2001-Dec2001	510	59	11.56%
3	Jan2002-Dec2002	613	56	9.13%
4	Jan2003-Dec2003	523	52	9.94%
5	Jan2004-Dec2004	469	37	7.88%
6	Jan2005-Sept2005	300	24	8.0%
			Average	9.75%

Table No I A shows the incidence of esophageal specimens received after initial endoscopic evaluation for management during the period from 2000 to 2005.

The average incidence is 0.30%

Table I A

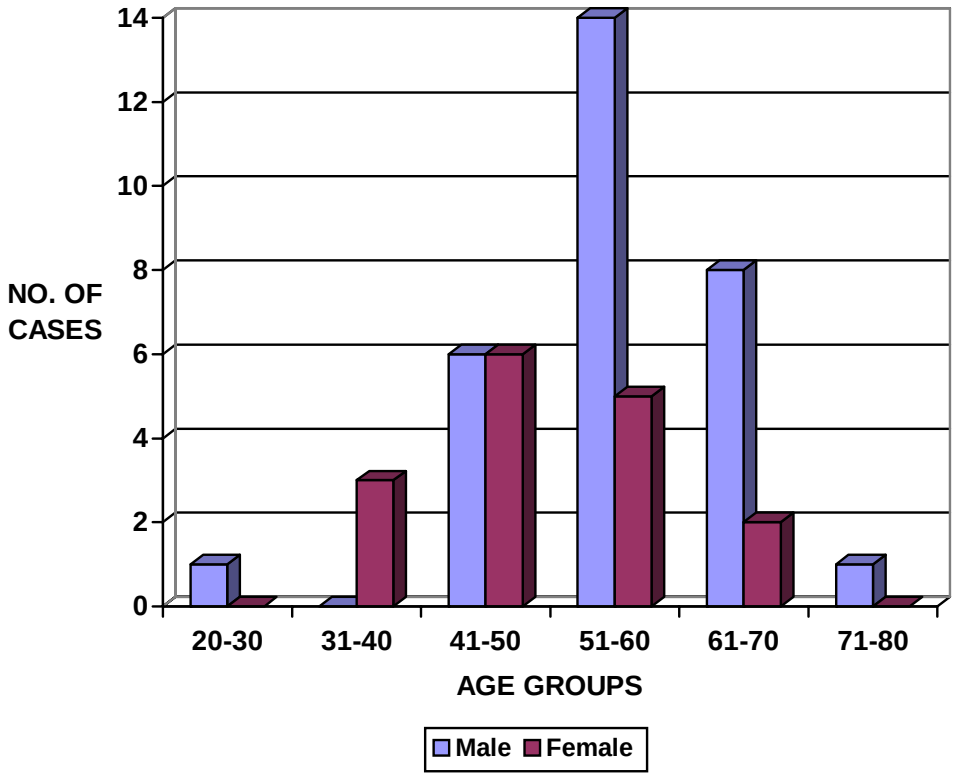
S. No	Period	Total no of specimens	Esophageal Specimens	Percentage
1	Jan 2000-Dec 2000	2124	4	0.188%
2	Jan- Dec 2001	2306	6	0.260%
3	Jan-Dec 2002	2609	6	0.229%
4	Jan-Dec 2003	2344	11	0.469%
5	Jan- Dec 2004	2795	16	0.572%
6	Jan-Sep 2005	2280	3	0.131%
			Average	0.308%

AGE INCIDENCE:

The patients clinically manifested with dysphagia, weightloss, nausea and vomiting who were further biopsied were divided into 6 groups according to their age [i.e. 20-30yrs, 31-40yrs, 41-50yrs, 51-60yrs, 61-70yrs, 71-80yrs.]

There was increased incidence of esophageal lesion observed in the age group of 51-60 yrs (33.21%) followed by 41-50 yrs (24.55%) and 61-70yrs (14.49%).

AGE AND SEX DISTRIBUTION OF RESECTED CASES



The age distribution of esophageal biopsies is given in Table No II

Similarly the age distribution of esophageal specimens received were also calculated accordingly as given in Table II A.

Table II

S. No	Age Group	No. of Cases			Percentage
		M	F	T	
1	20-30yrs	9	6	15	5.415%
2	31-40yrs	19	18	37	13.357%
3	41-50 yrs	42	26	68	24.548%
4	51-60yrs	73	19	92	33.2129%
5	61-70 yrs	43	11	54	19.495%
6	71-80yrs	7	4	11	3.97%

Table II A

S. No	Age Group	No. of Cases			Percentage
		M	F	T	
1	20-30	1	0	1	2.173%
2	31-40	0	3	3	6.521%
3	41-50	6	6	12	26.086%
4	51-60	14	5	19	41.304%
5	61-70	8	2	10	21.739%
6	71-80	1	0	1	2.173%

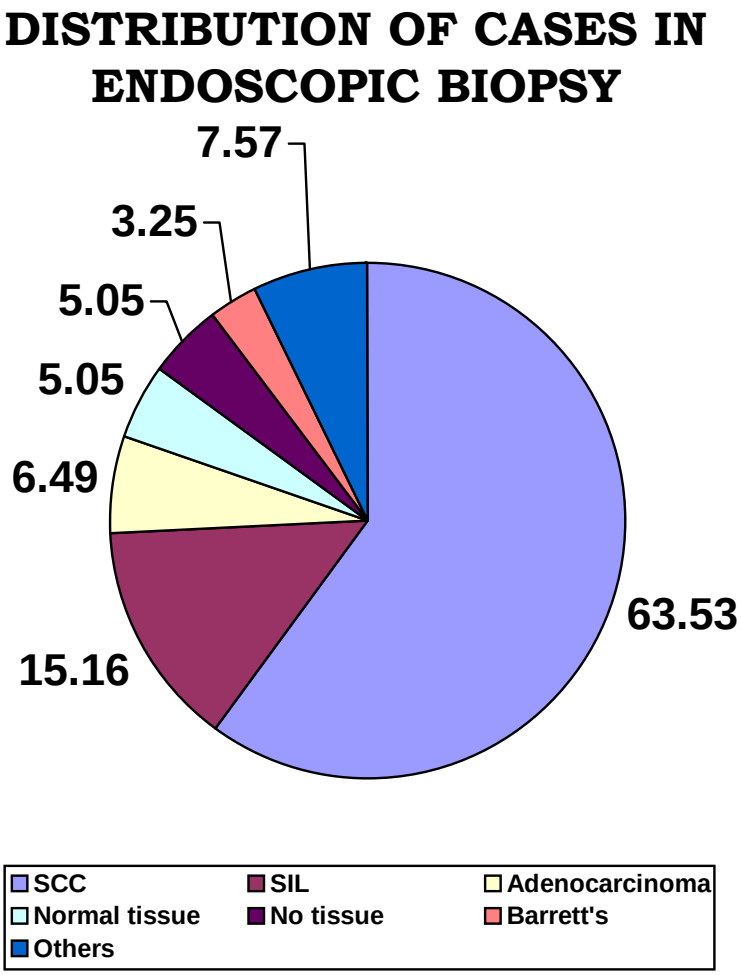
Most of the patients belong to lower socio economic status, lived in overcrowded surroundings

Table No III shows the distribution of cases according to their clinical presentation in endoscopic biopsies.

Table No III

S. No	Features	Number of Cases	Percentage
1	Dyspepsia	69	24.91%
2	Dysphagia	250	90.25%
3	Loss of weight	194	70.04%
4	Anorexia	152	54.87%
5	Vomiting	69	24.91%
6	Odynophagia	78	28.16%
7	Regurgitation of food	90	32.49%
8	Hoarseness of voice	2	0.72%
9	Heart burn	52	18.77%

Most of the patients in our study had complained of dysphagia (90.25%) followed by loss of weight (70.04%) and anorexia (54.87%) .


DISTRIBUTION OF CASES IN ENDOSCOPIC BIOPSY
7.57
3.25
5.05
5.05
6.49
63.53
15.16
SCC
SIL
Adenocarcinoma
Normal tissue
No tissue
Barrett's
Others

ESOPHAGEAL BIOPSY EVALUATION

277 cases of esophageal biopsy were received and histopathological evaluation was done.

Among the 277 cases, 9 cases were Barrett's (Fig 6) (3.249%), 18 cases were diagnosed as adenocarcinoma (Fig 7 & 8) (6.498%) 176 cases were diagnosed as squamous cell carcinomas (Fig 9) (63.18%) 42 cases were squamous intraepithelial lesion (Fig 10) (15.16%) 14 cases were interpreted as normal stratified squamous epithelium (5.05%) 14 cases were interpreted as only necrotic material /no tissue (5.05%). Table No IV shows the incidence of lesions and its percentage.

Table No IV

S. No	Type of lesions	Male	Female	Total	Percentage
1	NON NEOPLASTIC LESION Normal	11	3	14	5.05%
	No tissue	11	3	14	5.05%
2	BARRETT'S ESOPHAGUS	7	2	9	3.25%
3	NEOPLASTIC –BENIGN Squamous Papilloma	1	0	1	0.36%
	Fibromuscular Polyp	1	0	1	0.36%
4	PRENEOPLASTIC SIL	27	15	42	15.16%
5	MALIGNANT NEOPLASM Squamous cell carcinoma-Usual Type	115	60	175	63.18%
	Sarcomatoid Carcinoma	1	0	1	0.36%
	Adenocarcinoma	16	2	18	6.49%
	Adenosquamous Carcinoma	1	0	1	0.36%
	Malignant Melanoma	0	1	1	0.36%

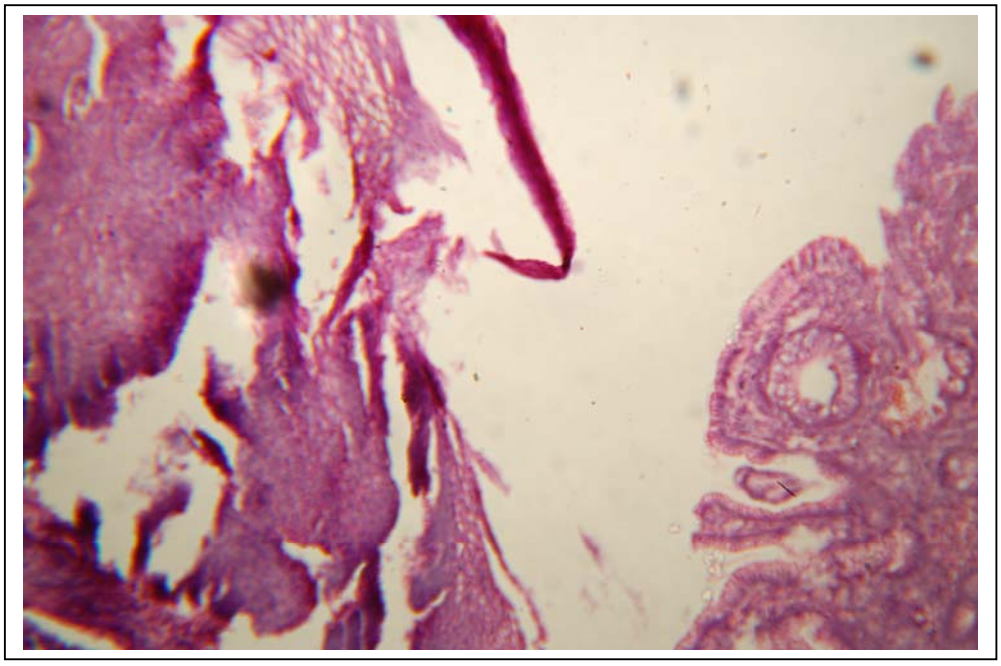


Fig 6 Barrett's esophagus –intestinal metaplasia with goblet cells between mucous producing cells (H & E X100).

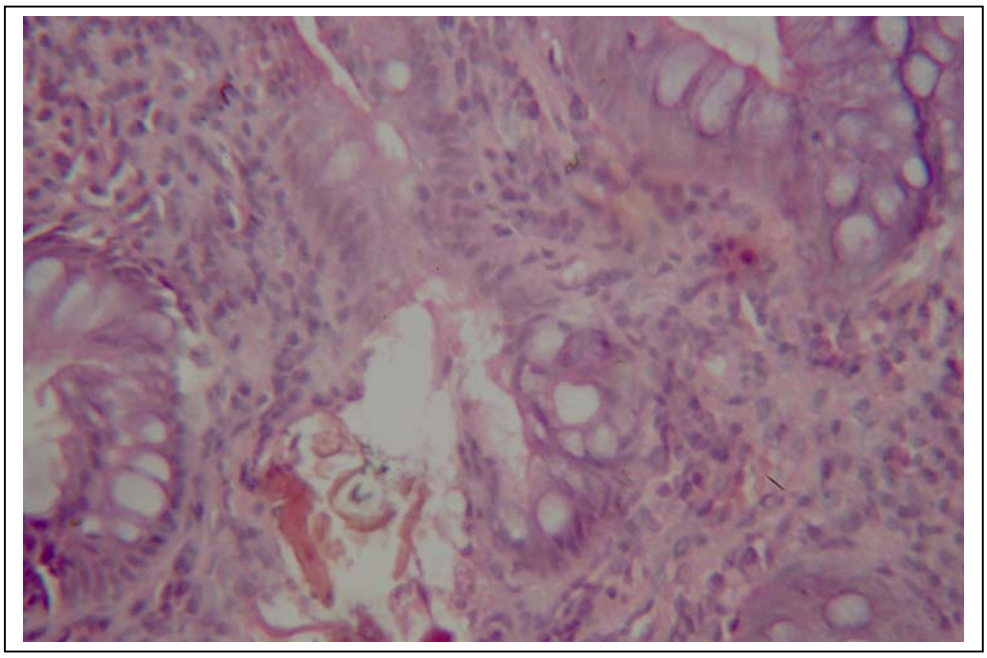


Fig 6A Barrett's esophagus –columnar epithelium with goblet cells (H & E X400).

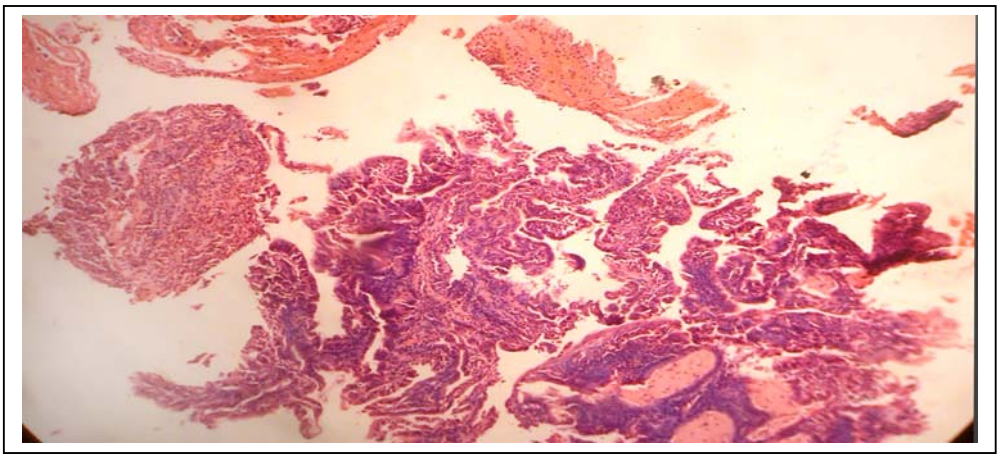


Fig 7 Adenocarcinoma arising from Barrett's esophagus –glandular and tubular profiles, lined by columnar cells. Biopsy (H & E X100).

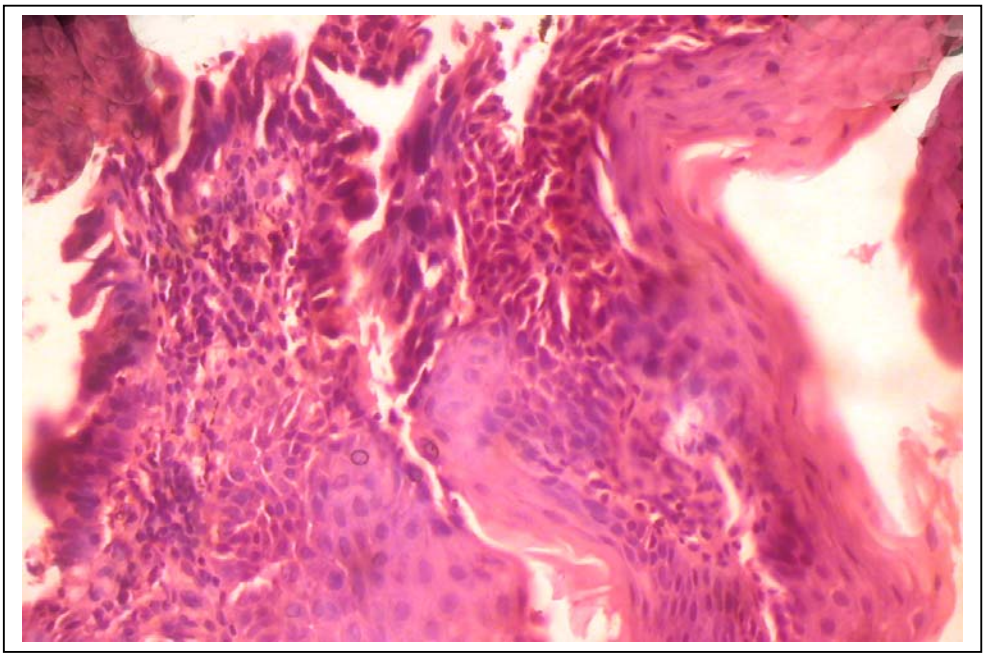


Fig 7A Squamous epithelium continuous with neoplastic glandular epithelium high power view of above X400↑.

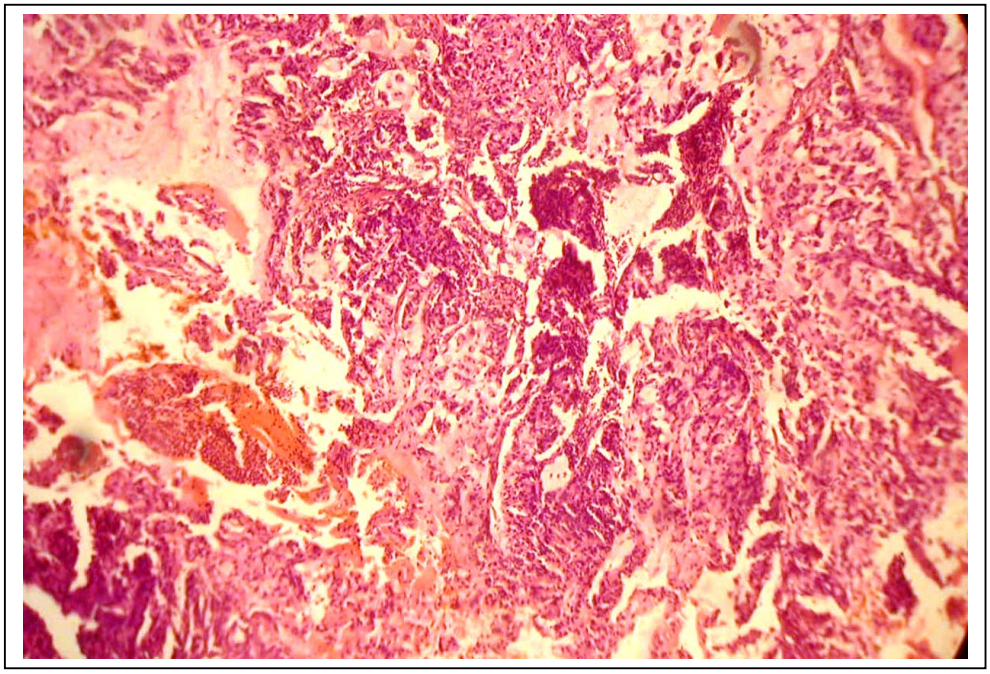


Fig 8 Mucoid carcinoma –abundant mucin pool with signet ring cells. Biopsy (H & E X100).

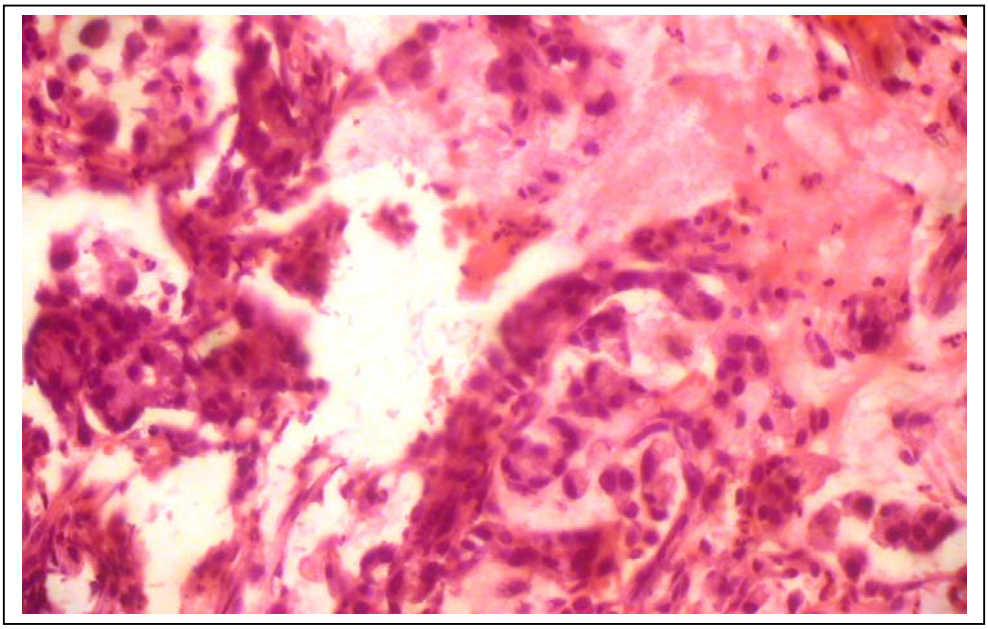


Fig 8A Mucoid carcinoma clusters of neoplastic cells against pool of mucin. Biopsy (H & E X400).

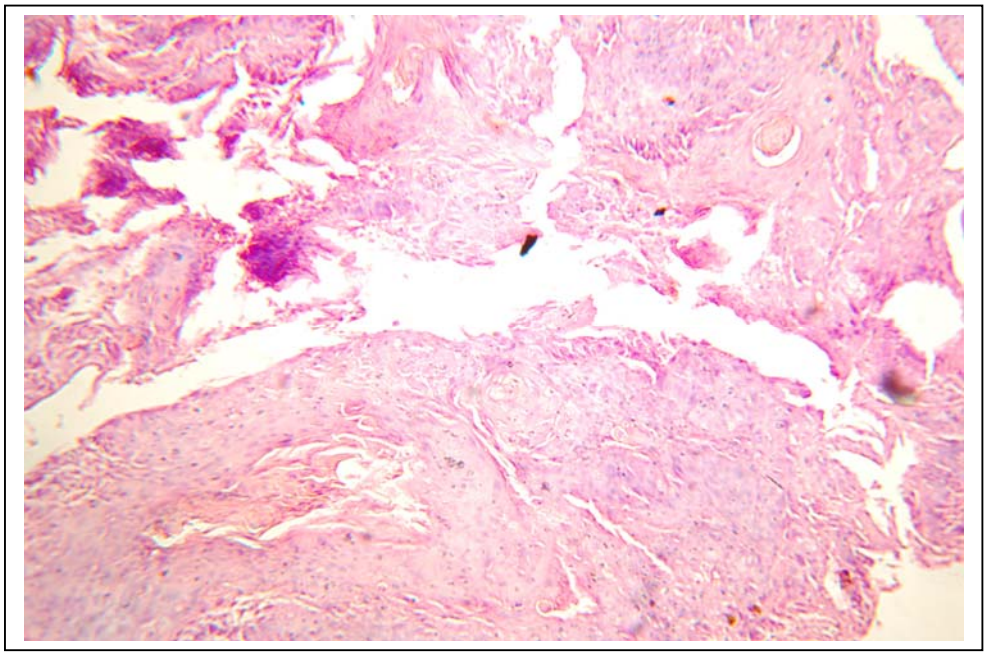


Fig 9 Well differentiated squamous cell carcinoma – Keratin pearl formation.Biopsy (H & E X 100).

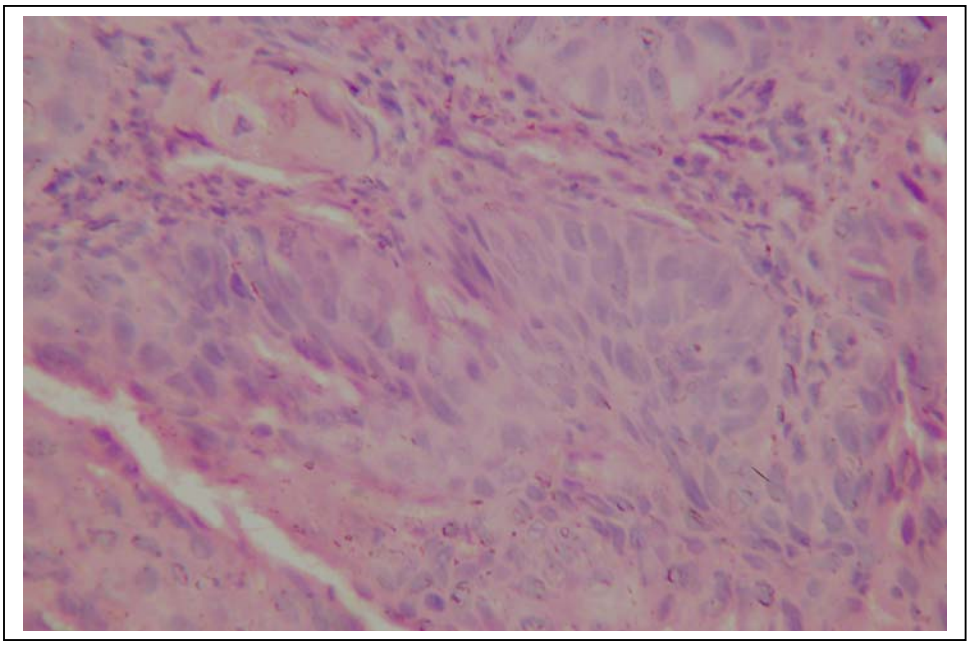


Fig 9A High power view of above X 400↑.

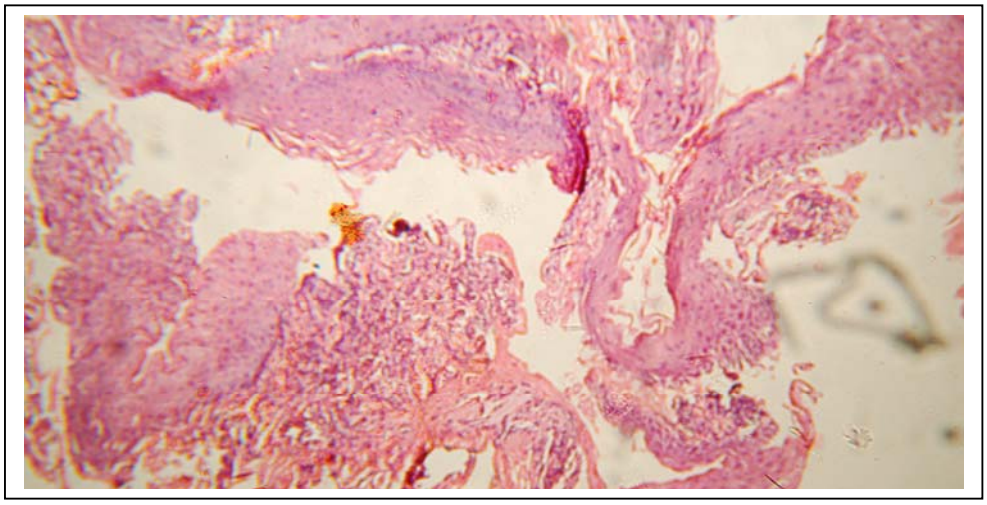


Fig 10 Low grade squamous Intraepithelial lesion dysplastic changes confined to the lower one third of mucosa. Biopsy (H & E X 100).

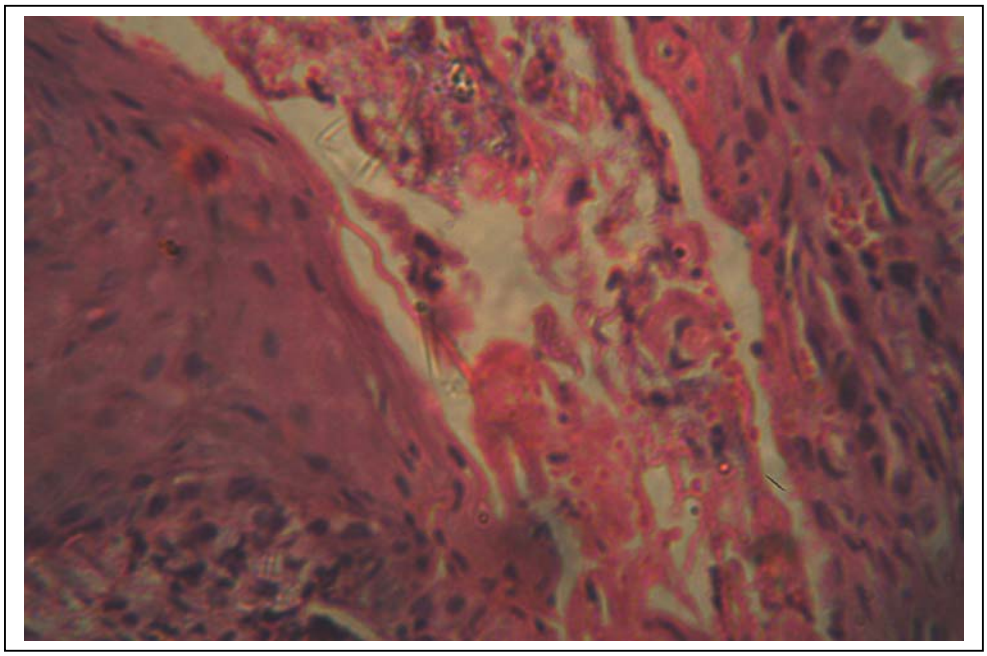


Fig 10A High power view of above X400↑.

Table no IV shows Squamous cell carcinoma (SCC) of the esophagus is the commonest malignant neoplasm [176 cases, (63.54%)] followed by SIL [42 cases, (15.16%)]. Incidence of Adenocarcinoma esophagus is only 6.49% Table IV.A and Table IV B shows, in esophageal biopsies Squamous cell carcinoma was the commonest neoplasm (males 41.87%, Females 21.66%) followed by squamous intra epithelial lesion (Males 9.75%, Females 5.42%) Incidence of Barrett's esophagus characterised by metaplastic columnar epithelium intestinal type with goblet cells that has replaced the original squamous lined mucosa is also observed. In males 7 cases of BE (2.53%) was observed and 16 cases of Adenocarcinoma (5.78%) was seen. Table IV A and B also clearly shows the esophageal lesion (Neoplastic, pre neoplastic) are common in males when compared with females and the incidence of Adenocarcinoma is much low when compared with classical SCC.

Table IV A

Male Incidence

S. No	Type of Lesion	No. of Cases	Percentage
1	Barrett's esophagus	7	2.527%
2	SIL	27	9.75%
3	SCC	116	41.877%
4	Adenocarcinoma	16	5.776%

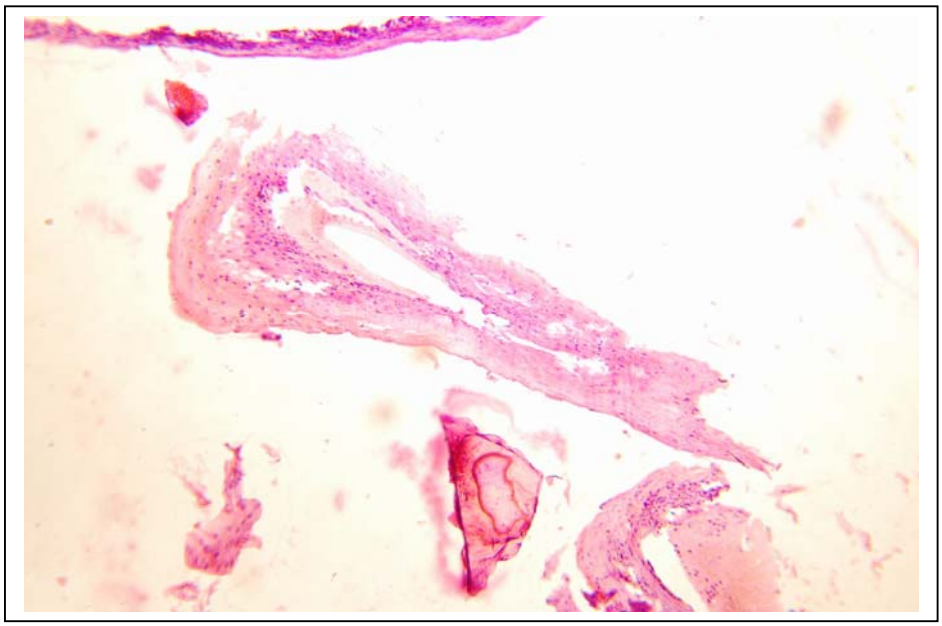


Fig 11 Squamous papilloma –stratified squamous epithelium with central fibrovascular core. Biopsy (H & E X100).

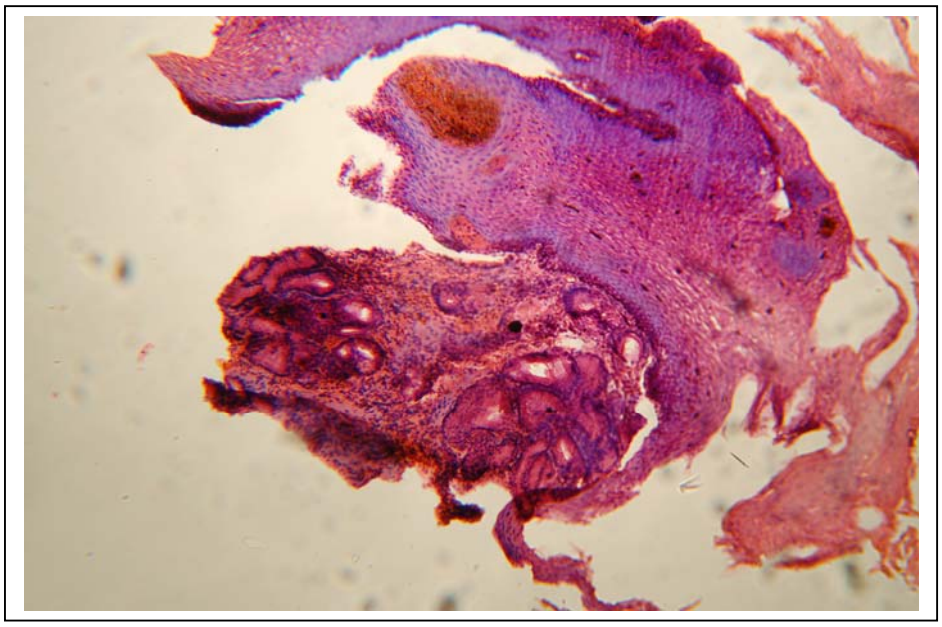


Fig 12 Fibrovascular polyp – intact surface epithelium with loose connective tissue in lamina propria. Biopsy (H & E X 100).

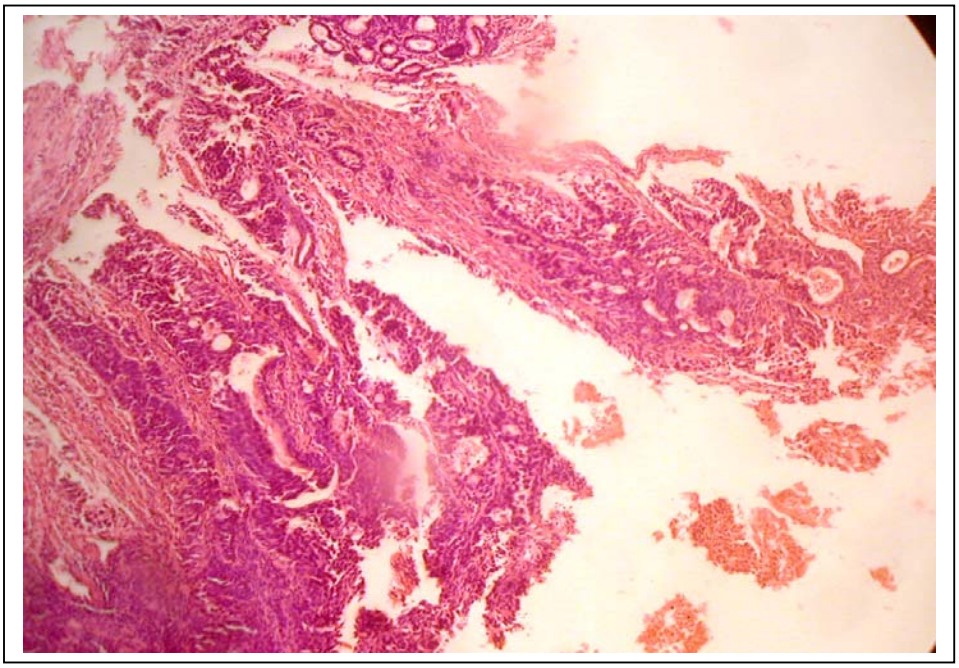


Fig 13 Adenosquamous carcinoma –mixed squamous and glandular differentiation of neoplastic cells. Biopsy (H & E X100).

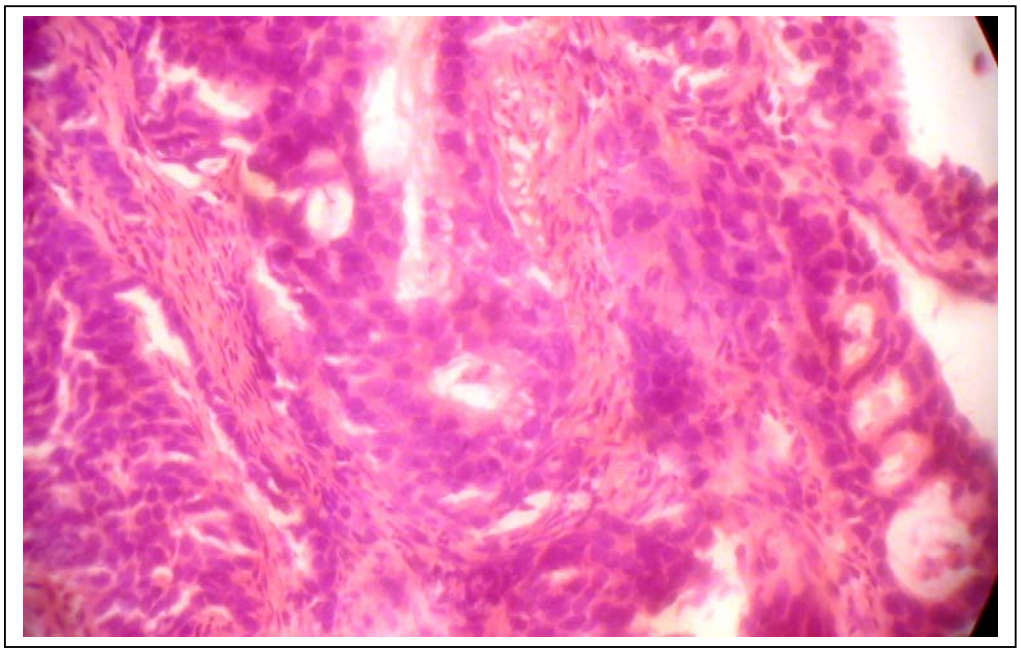


Fig 13A High power view of above X400 –with glandular component. ↑.

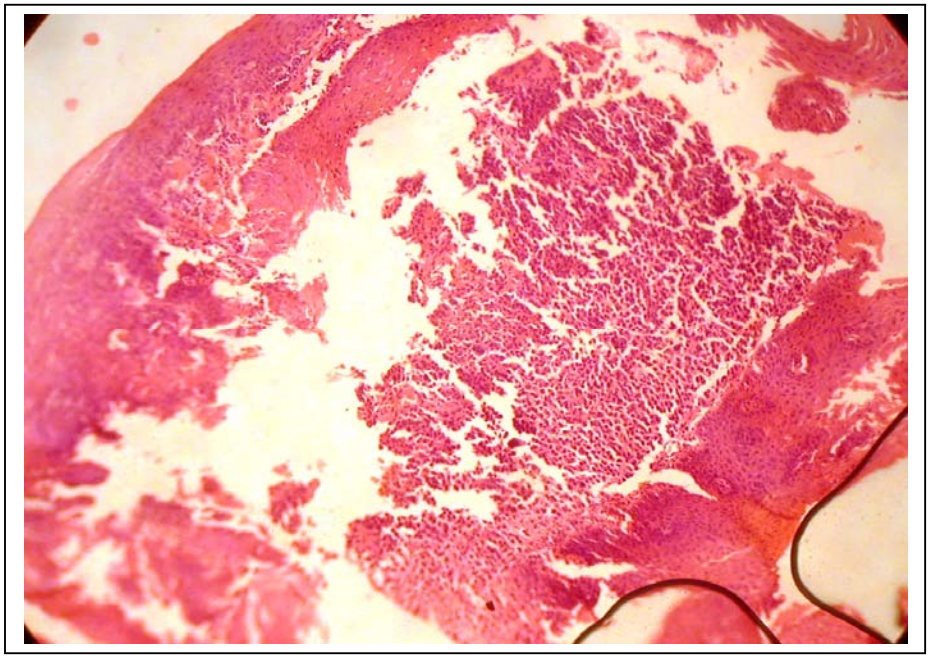


Fig 14 Malignant melanoma –basal proliferation of melanocytes and invasive epitheloid component. Biopsy (H & E X100).

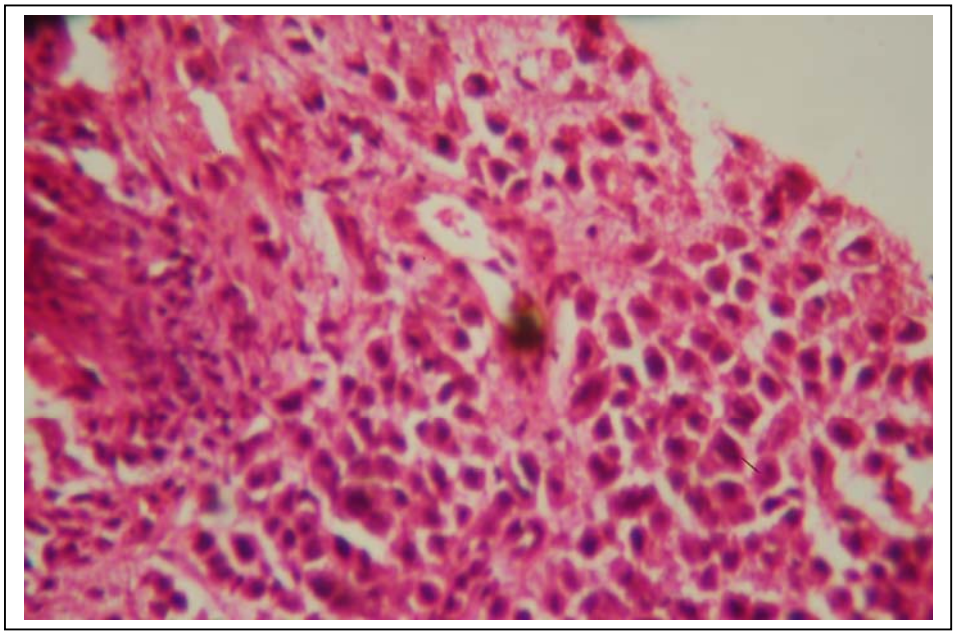


Fig 14A Malignant melanoma with obvious intra cytoplasmic pigmentation. (H & E X400).

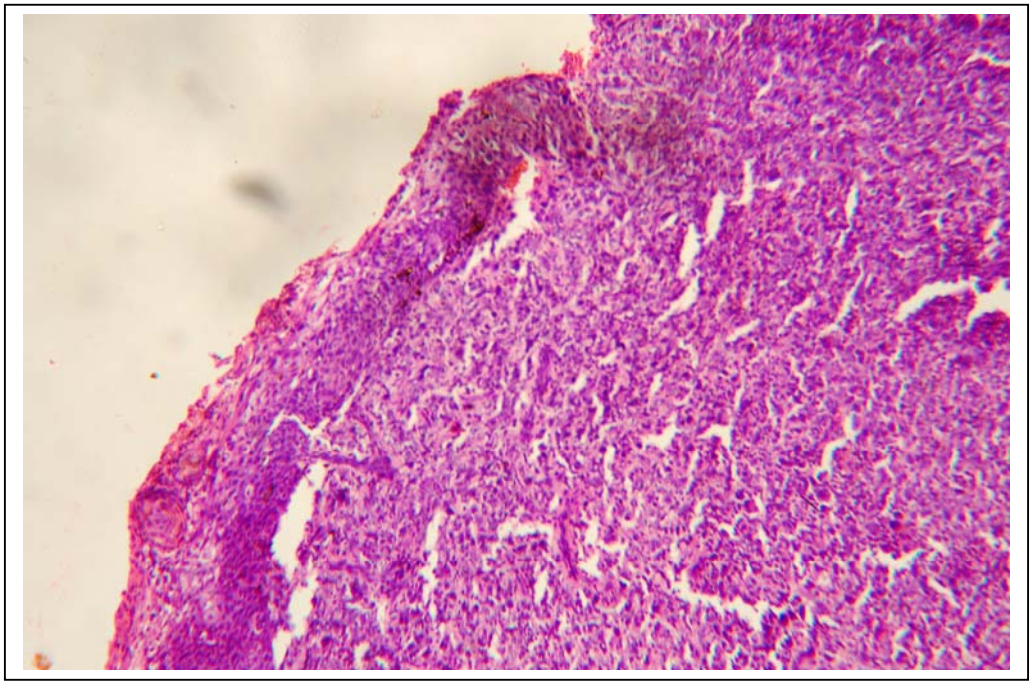


Fig 15 Sarcomatoid carcinoma –Epitheloid malignant squamous cells blend imperceptibly with an undifferentiated sarcomatous component. Biopsy (H & E X100).

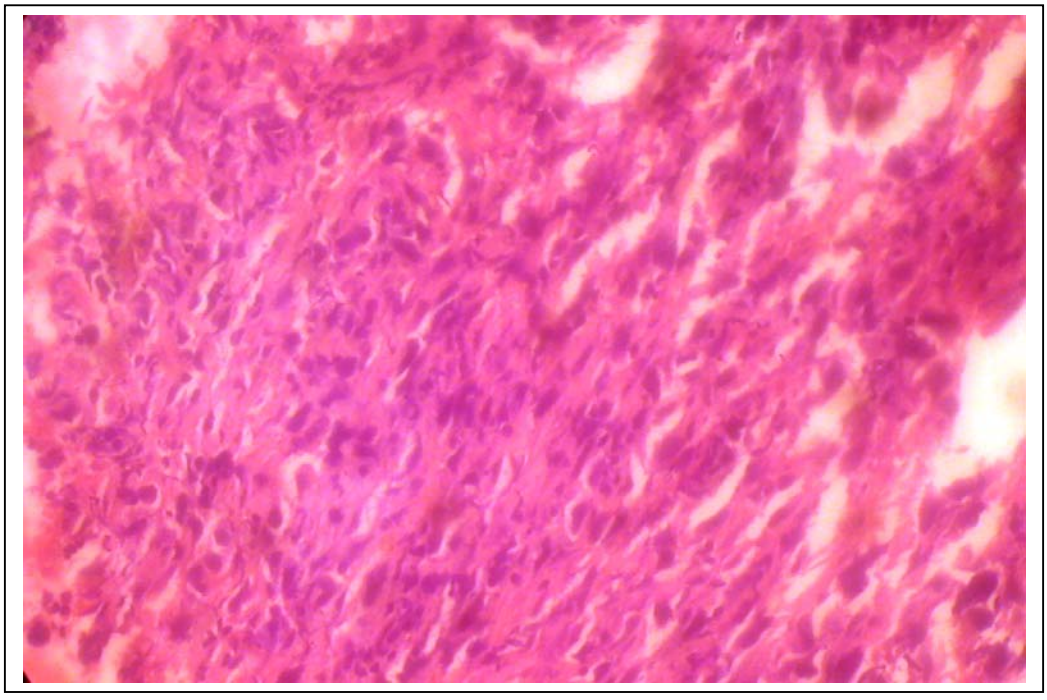


Fig 15A High power view of above X400 –proliferation of spindle cells with hyperchromatic elongated nuclei. ↑.

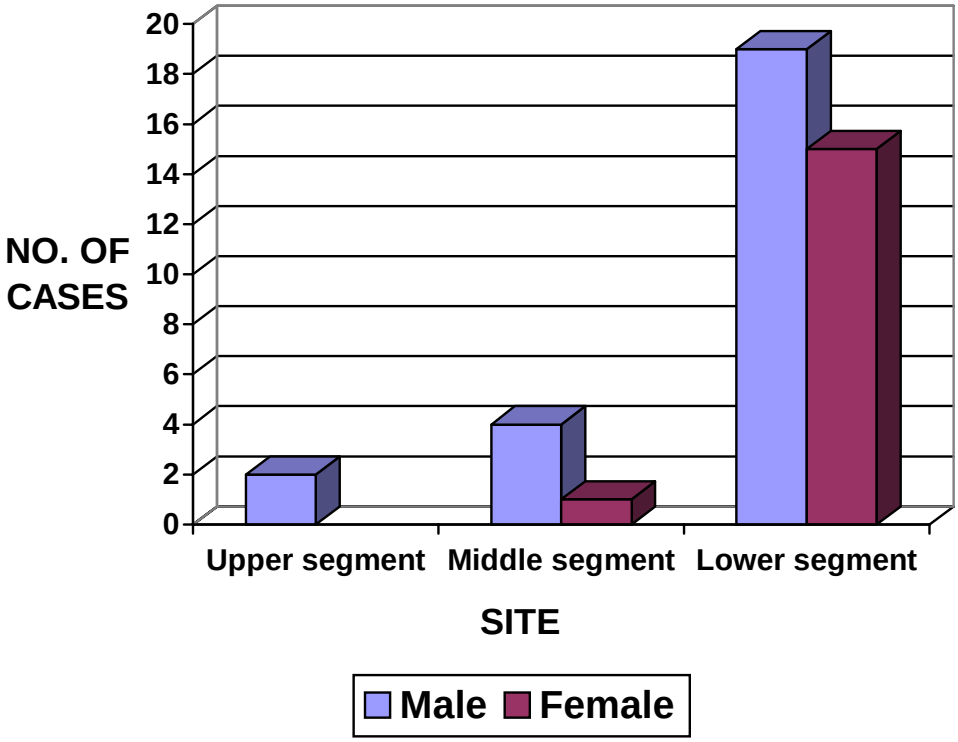
Table IV B

Female Incidence

S. No	Type of Lesion	No. of Cases	Percentage
1	Barrett's esophagus	2	0.72%
2	SIL	15	5.415%
3	SCC	60	21.66%
4	Adenocarcinoma	2	0.722%

Among the 277 cases one case of Squamous Papilloma (Fig 11) which occurred in a 50 yrs male with characteristic features of central core of connective tissue covered with hyperplastic Squamous cells in an orderly arrangement and without dysplastic changes was observed. One case of fibromuscular polyp (Fig 12) composed of mesenchymal tissue which was fibrous and covered by intact Squamous epithelium occurred in 38 yrs males. One case of adenosquamous carcinoma (Fig 13) characterised by proliferation of malignant glands adjacent to malignant squamous epithelium was observed in 70 yrs male. One case of malignant melanoma (Fig 14) characterised by small dark, flat lesion in the upper GI endoscopy which revealed aggregates of pleomorphic cells with hyperchromatic nuclei, binucleated cells and intracellular melanin pigmentation, was observed in 60 yrs female. One case of sarcomatoid carcinoma (Fig 15) with features of both spindle cells and squamous cell carcinoma components was observed in 55 yrs male.

SITE WISE DISTRIBUTION OF RESECTED SPECIMEN



HISTOPATHOLOGICAL EVALUATION:

46 esophagectomy specimens were received, of which 41 cases (89.13%) were squamous cell carcinoma and 5 cases were adenocarcinoma (10.86%).

The received specimens were categorised according to the site of presentation whether in upper 1/3rd, middle 1/3rd and lower 1/3rd and also categorized according to the morphological appearance.

Table No V shows the distribution of cases according to the morphological appearance.

Table No V

S. No	Type of Growth	Number of Cases	Percentage
1	Proliferative / Fungating	39	84.78%
2	Ulcerative	4	8.69%
3	Infiltrative	3	6.52%

Most of the presented cases were in advanced stage with exophytic fungating growth on gross appearance (Fig 16,17).

Most of the cases presented in lower 1/3rd segment of esophagus (34 cases 73.91%). Relative distribution of squamous cell carcinoma according to site is shown in Table No V A.

Table No V A

S. No	Site	Number of Male cases	Percentage	Number of Female Cases	Percentage	Total	Percentage
1	Upper 1/3rd	2	4.35%	-	-	2	4.35%
2	Middle 1/3rd	4	8.69%	1	2.17%	5	10.86%
3	Lower 1/3rd	19	41.305	15	32.60%	34	73.91%

Table No V B shows the relative distribution of squamous cell carcinoma in both sex.

Table No V B

Male	Number of cases 25	60.97%
Female	Number of cases 16	39.03%

Majority of patients who had squamous cell carcinoma were males (60.97%).

Table No V C shows the relative distribution of squamous cell carcinoma according to differentiation.

Table No V C

S. No	Degree of differentiation	Number of cases		Total	Percentage
		Male	Female		
1	Well differentiated SCC	19	7	26	63.41%
2	Moderately differentiated SCC	8	5	13	31.707%
3	Poorly differentiated SCC	2	0	2	4.88%

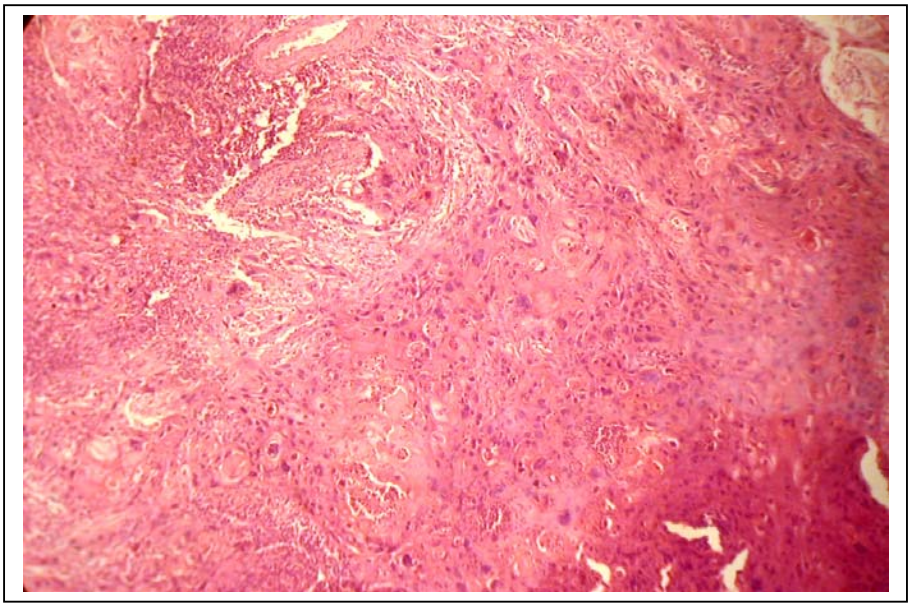


Fig 18 Well differentiated squamous cell carcinoma –Keratin pearl formation and intercellular bridges (H & E X100).

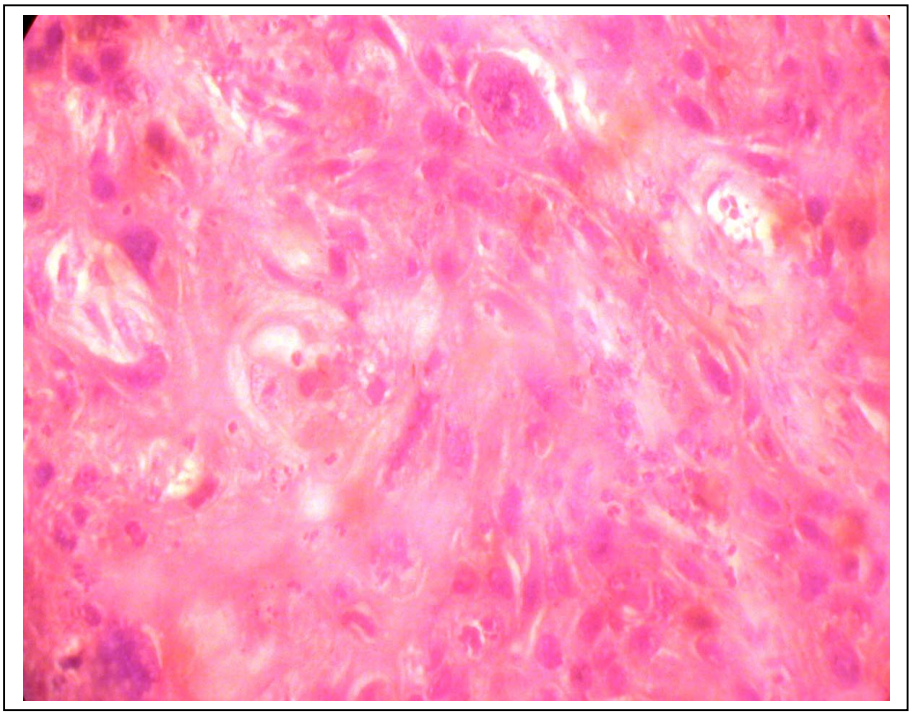


Fig 18A High power view of above X400 ↑.

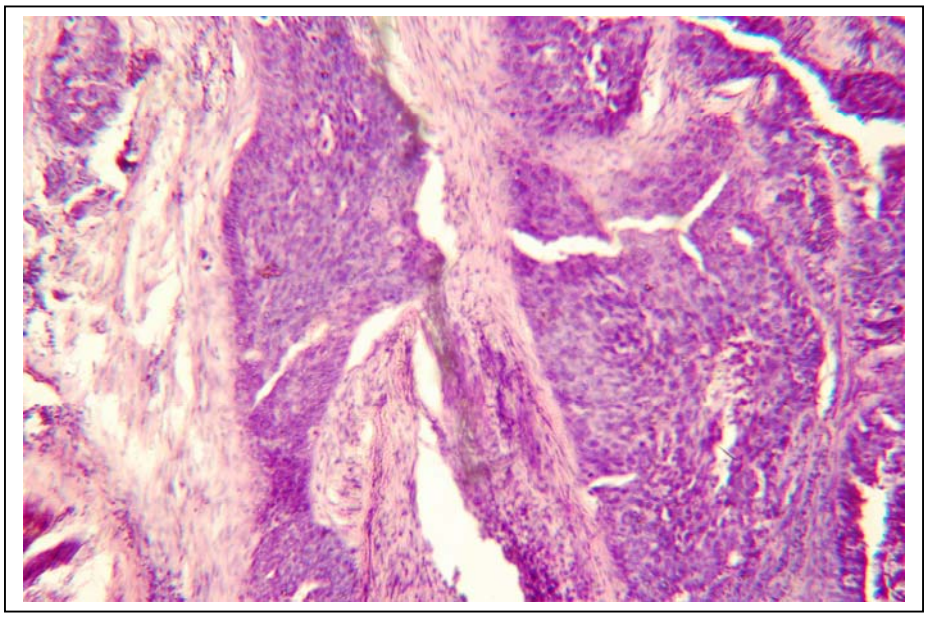


Fig 19 Moderately differentiated SCC – with intercellular bridges (H & E X400).

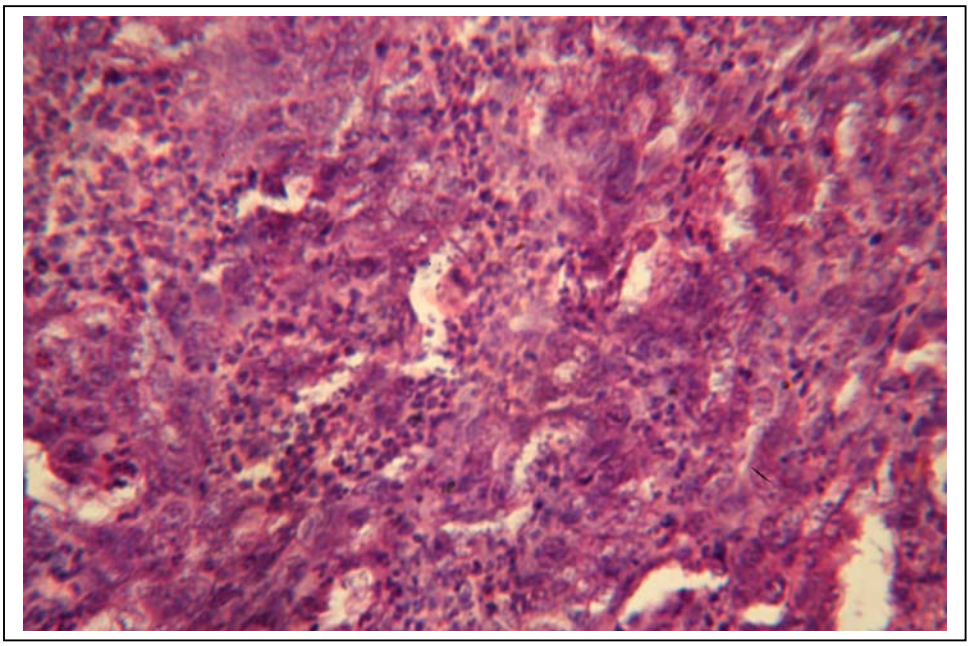


Fig 20 Poorly differentiated SCC –large pleomorphic cells with high nuclear cytoplasmic ratio with brisk lymphocytic response.(H &E X400).

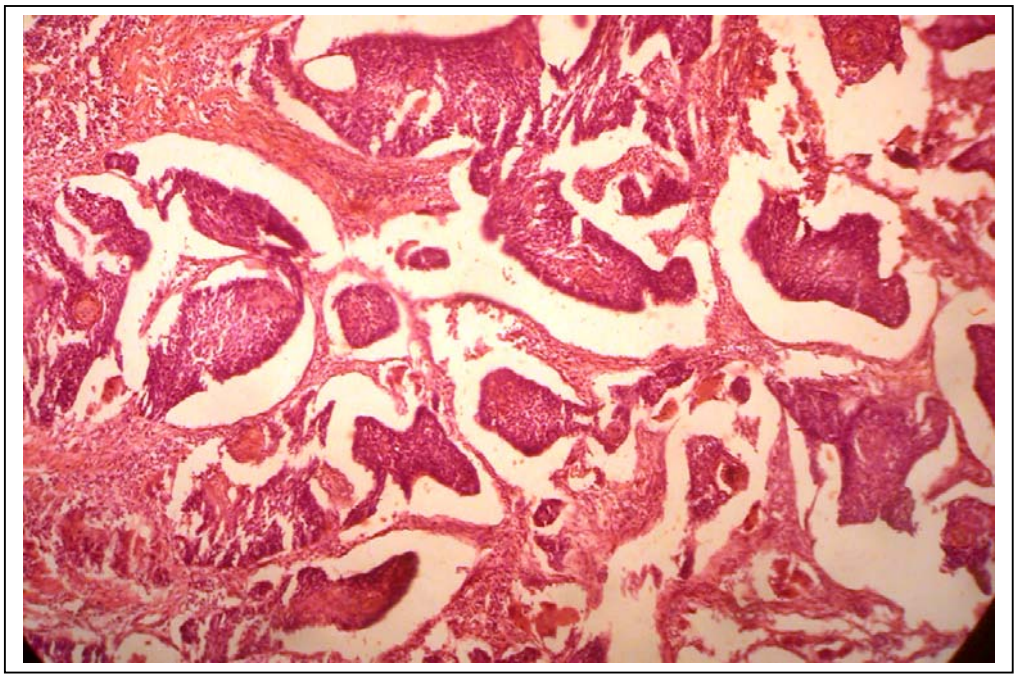


Fig 21 SCC with basisquamous features -basal cells with squamous differentiation and retraction artefact (H & E X100).

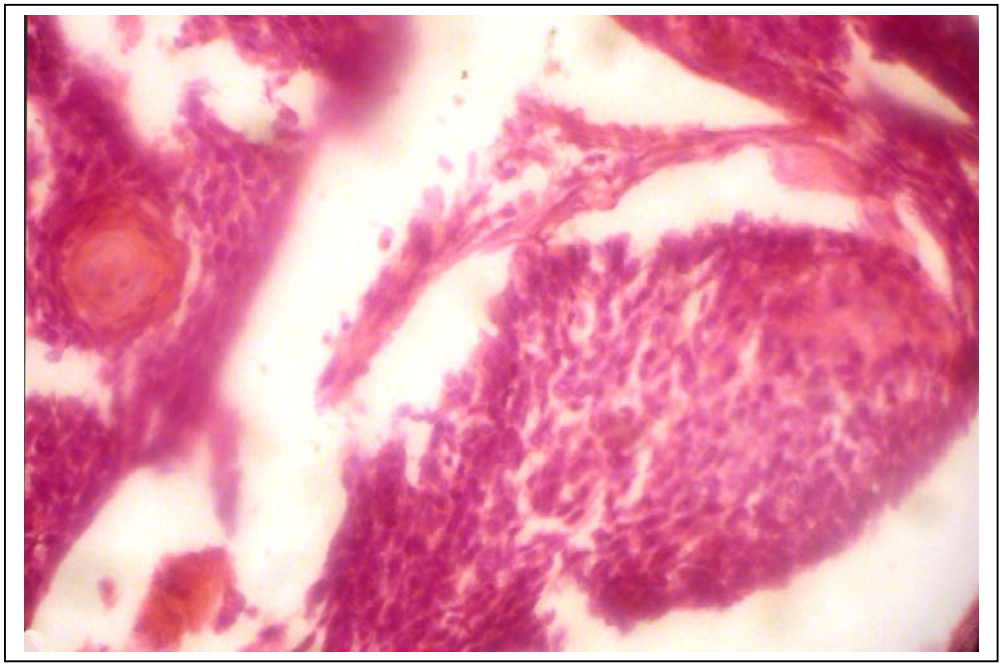


Fig 21A High power view of above X400 with Keratocysts. ↑.

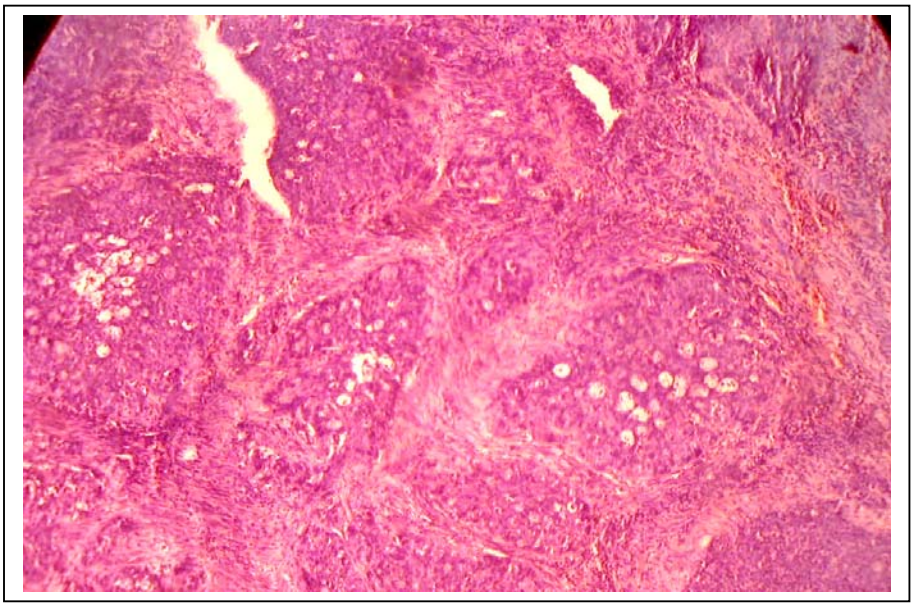


Fig 22 Basaloid carcinoma –islands of basaloid cells with peripheral palisading and cribriform pattern (H & E X100).

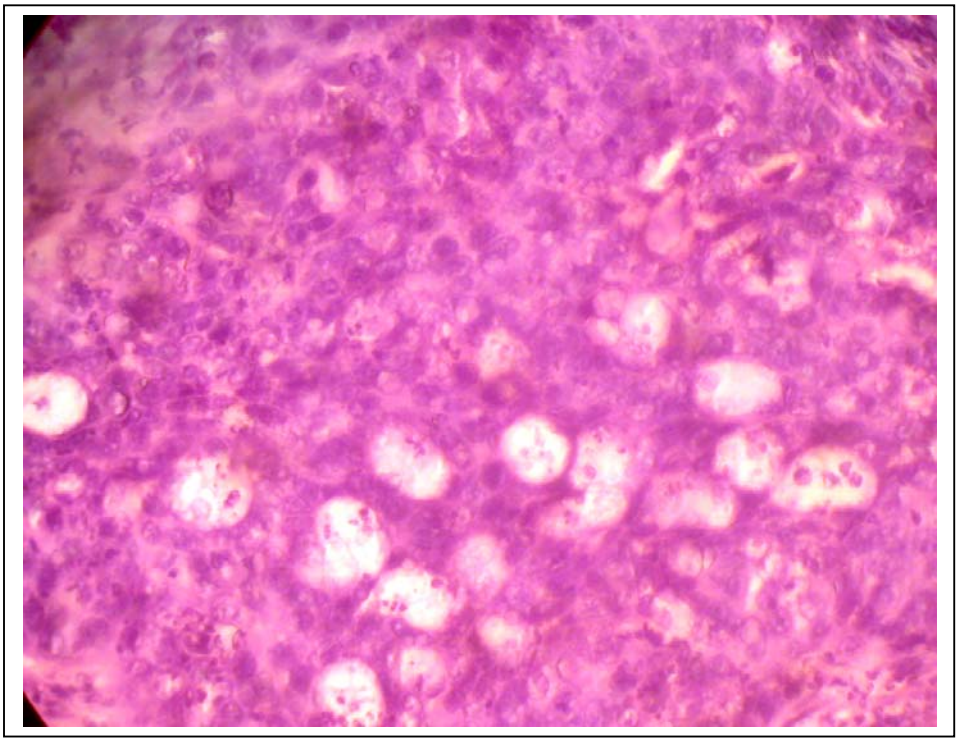


Fig 22A Basaloid carcinoma – adenoid cystic spaces containing mucoid hyaline like material (H & E X400).

Most of the adenocarcinomas occurred in males Table No VI shows the relative distribution of adenocarcinoma according to differentiation.

Table No VI

S. No	Degree of differentiation	Number of cases		Total	Percentage
		Male	Female		
1	Well differentiated adenocarcinoma	3	0	3	60%
2	Moderately differentiated adenocarcinoma	1	0	1	20%
3	Poorly differentiated adenocarcinoma	1	0	1	20%

Most of the squamous cell carcinoma were well differentiated (26 cases –63.41%) (Fig 18).

13 cases of squamous cell carcinoma were moderately differentiated (Fig 19) (13 cases-31.71%).

Only 2 cases of squamous cell carcinoma were poorly differentiated (Fig 20) (4.88%).

One poorly differentiated squamous cell carcinoma case had areas of basisquamoid picture and focal spindling. One moderately differentiated squamous cell carcinoma case had areas of basisquamous features. (Fig 21) One of the esophagectomy specimen had features of basaloid carcinoma (Fig 22) characterised by proliferation of oval to round, large pleomorphic basaloid cells with open pale chromatin pattern, small nuclei and scant cytoplasm arranged in solid and cribriform lobules often showing central necrosis, nests of tumor cells show peripheral palisading.

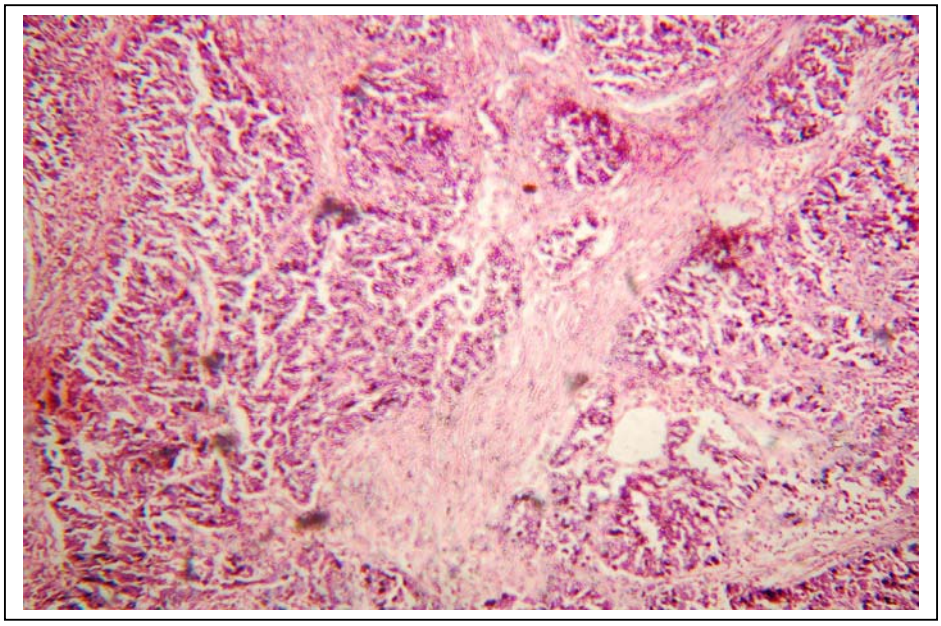


Fig 23 Poorly differentiated adenocarcinoma –diffuse infiltration of neoplastic cells, with desmoplastic stroma (H & E X100).

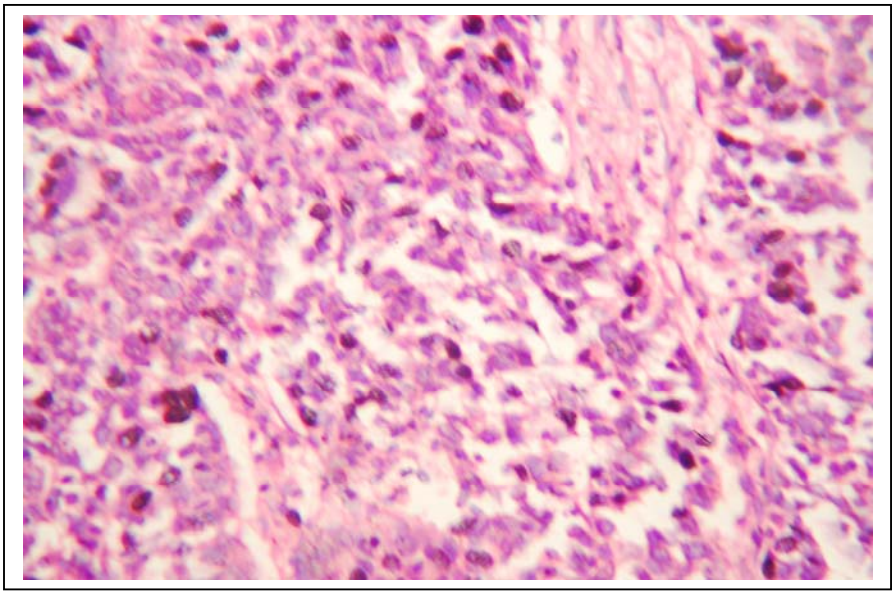


Fig 23A High power view of above X 400↑.

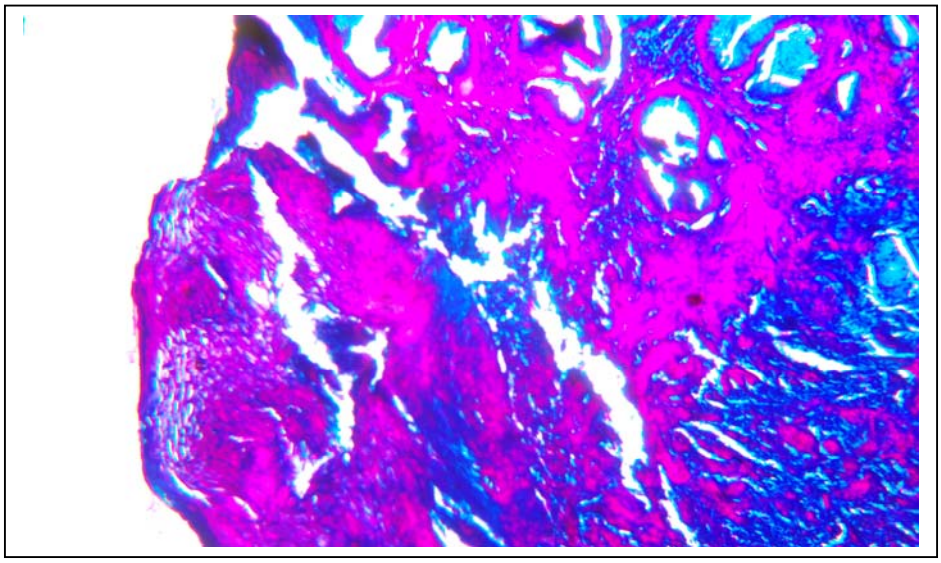


Fig 24 Barrett's esophagus – Alcian blue (2.5) with PAS Positivity.

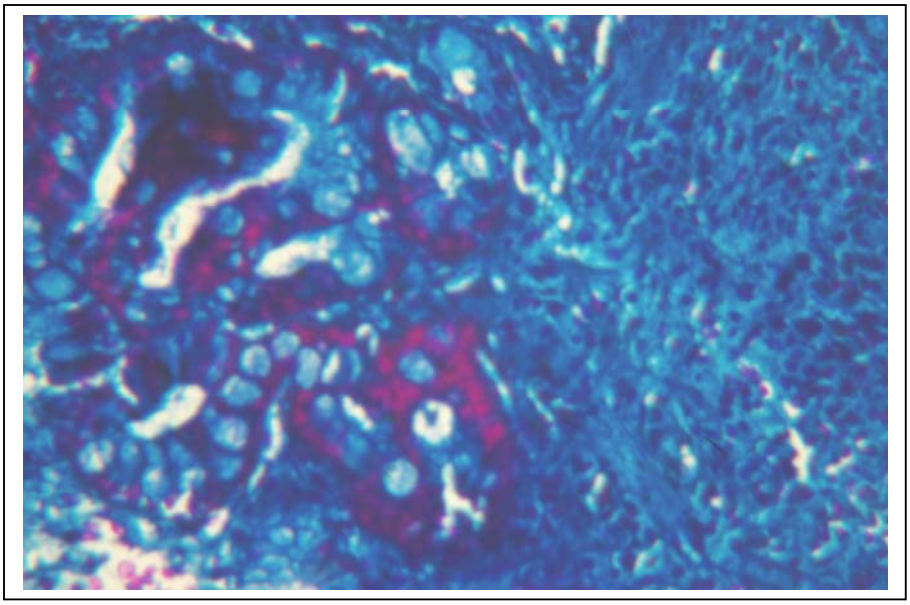


Fig 24A High Power view of above X400- scattered intensly blue staining goblet cells of Barrett's metaplastic epithelium ↑.

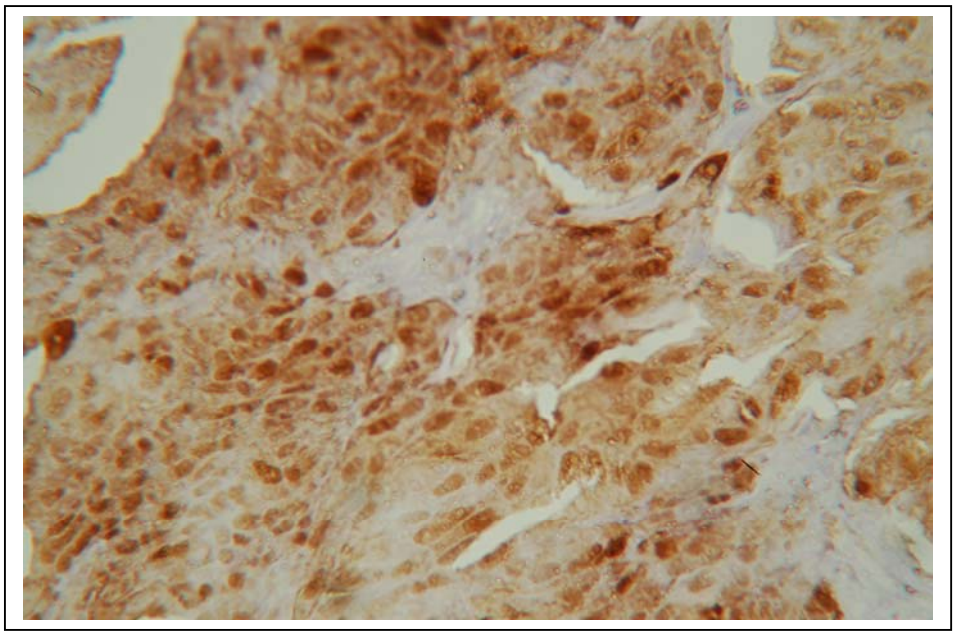


Fig 25 Intense nucleocytoplasmic immunoreactivity for S100 protein in malignant melanoma X400.

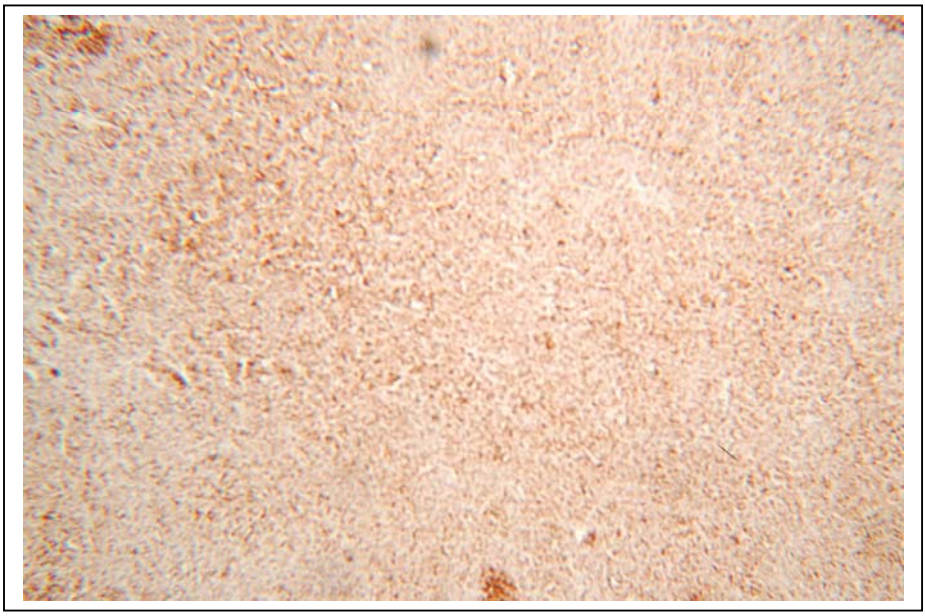


Fig 26 Sarcomatoid carcinoma exhibiting intense vimentin positivity.X100.

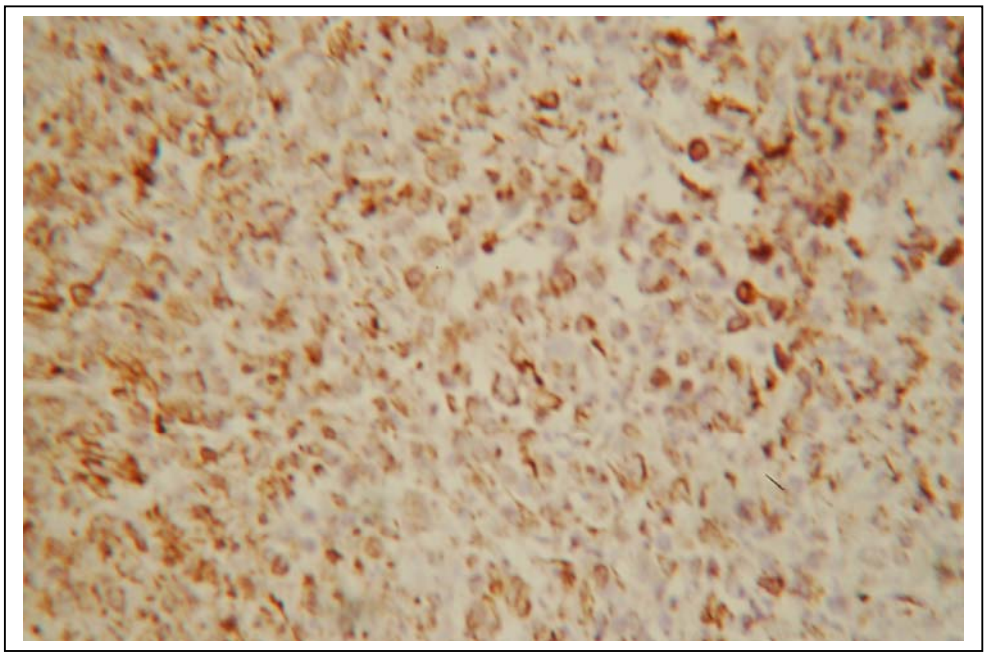


Fig 26A High power view of above X400↑.

Most of the adenocarcinomas were well differentiated except for two, one was moderately differentiated and the other Poorly differentiated. (Fig 23)

In Barrett's esophagus histochemistry with Alcian blue PAS was performed (Fig 24). Immunohistochemistry was performed in 2 doubtful cases and the results are shown in Table No VII. (Fig 25, 26)

Table No VII

S. No	Cases	S100	Vimentin	Cytokeratin	EMA
1	Sarcomatoid Carcinoma	-	+	-	-
2	Malignant melanoma	+	-	-	-

DISCUSSION

The present study as in the others and literature shows that esophageal lesions particularly malignant neoplasms are one of the commonest cancers occurring in the world.

Barrett's esophagus, as a result of GERD is now an accepted cause for further adenocarcinoma at lower end of esophagus. The following Table No VIII shows the incidence of esophageal neoplasm in our institution.

Table No VIII

S. No	Year	Total No of Cancer patients	Esophageal Carcinomas			Percentage
			Male	Female	Total	
1	Jan 2000 to Dec 2000	397	37	4	41	10.327%
2	Jan 2001 to Dec 2001	426	36	6	42	9.859%
3	Jan 2002 to Dec 2002	511	47	6	53	10.371%
4	Jan 2003 to Dec 2003	423	32	11	43	10.165%
5	Jan 2004 to Dec 2004	385	24	16	40	10.389%
6	Jan 2005 to Sep 2005	320	18	3	21	6.562%

Between January 2000 and September 2005, 2462 cancer patients attended our institution and of them, 240 patients had esophageal cancers, which constitutes 9.748% of the cancer cases registered in our hospital.

The incidence of esophageal cancers is uniformly high in India with Kashmir falling in the Asian esophageal cancer belt having a maximum incidence of 43.6/lakh population and Bombay having an incidence of 15.2/lakh population.

The case of cancer in the Eastern and Northern parts, is thought to be due to widely prevalent habit of drinking large quantity of scorching hot tea. According to Dr K Met and Bhansali SK, low rainfall and high salinity in the soil are also related to the increased incidence of esophageal cancers in Coastal Orissa and Karnataka.

In the present study, as in the others, the incidence of esophageal lesions is high in the age group of 51-60 yrs followed by 41-50yrs. Das et al 1970 had also reported a high incidence of esophageal cancers in this age group. In contrast, this age incidence is considerably lower than that reported in the western countries, where it is common in sixtees and seventees.

In our study esophageal carcinomas occur more commonly in males, with male, female ratio of 8:1 in contrast with figures of Registrar-General of UK statistics of 1.5:1.0.

In our study 90% of cases complained of dysphagia and most of them had Grade III or Grade IV dysphagia. About 70% of patients had

loss of weight and anorexia followed by regurgitation of food in 33% of cases.

The figures come in well correlation with clinical evaluation conducted by Orringer et al and Sriram et al in Thanjavur March 2000, as shown in Table No IX

Table No IX

S. No	SYMPTOMS	ORRINGER (100 Cases)		SRIRAM (82 Cases)		PRESENT STUDY	
		No	%	No	%	No	%
1	Dysphagia	90	90%	82	100%	250	90.20%
2	Odynophagia	50	50%	26	31.7%	78	28.16%
3	Anorexia	48	48%	49	60%	152	54.87%
4	Regurgitation	30	30%	30	36.6%	90	32.49%
5	Hoarseness of voice	4	4%	3	3.6%	2	0.72%

In our study 2 cases of well differentiated squamous cell carcinoma esophagus is seen in 20 yrs of age, clearly shows the role of environment, food habits, nutrition and stress in the emergence of malignant neoplasm at the younger age group.

Esophageal cancer is modulated by not one single factor, but rather a large number of factors such as smoking, alcohol drinking and food consumption[10]. The particular contributions of these factors to esophageal cancer may differ among areas or countries along with variation in exposure levels, both individually and in combination.

Smoking has been proved to be a major risk factor for several sites cancer such as nasal, oral and lung cancers.

Ethanol is not a carcinogen itself, but may promote carcinogenesis[10] via, 1) Generation of free radical products during its metabolism, 2) solvent effects on tobacco and other carcinogens, 3) Induction of microsomal enzymes involved in carcinogen metabolism, 4) Metabolism to acetaldehyde which has proven to be a carcinogen in animal experiments.

Since the alcohol consumption is different among areas of India and the levels of other risk factors also vary, alcohol has a different contribution to esophageal cancer among various areas of India.

Furthermore, eating food rapidly increased the risk, while frequent consumption of fresh fruit, fresh vegetables and eggs was associated with protection. Foods not chewed adequately could disrupt the mucosal lining of carcinogenesis and growth of lesions.

Nutritional factors have been implicated in the etiology of esophageal cancer. Evidence from epidemiological and experimental studies suggests that deficiency of micronutrients[39] namely vitamin A, Zinc, Selenium and magnesium are associated with esophageal cancer.

Risk factor for squamous cell carcinoma of the esophagus have been identified such as tobacco, alcoholism, malnutrition, infection with

human papilloma virus. The risk factors associated with esophageal adenocarcinoma are less well defined.

The most important epidemiological difference between squamous cell carcinoma and adenocarcinoma however, is the strong association between gastroesophageal reflux disease and adenocarcinoma[2]. The results of a population based controlled study suggest that symptomatic gastroesophageal reflux is a risk factor for esophageal adenocarcinoma.The frequency, severity and duration of reflux symptoms were positively associated with increased risk of adenocarcinoma.

Barrett's esophagus is noted in 3% to 12% of patients with symptomatic GERD who undergo endoscopy with biopsy. The definition of Barrett's esophagus has been refined over the past 10 years. In studies published in the 1970s and 1980s,Barrett's esophagus was commonly defined by the presence of atleast 2 to 3 cm of columnar epithelium in the lower esophagus. This requirement avoided the diagnostic difficulties posed by the 'normal' occurrence of cardiac or fundic type epithelium in the distal esophagus. However more recent studies have found that only those patients with intestinal metaplasia, defined by the presence of acid mucin containing goblet cells, are at increased risk of developing dysplasia and adenocarcinoma, and as such,

the definition of Barrett's esophagus has now become dependent upon identification of goblet cells.

Recently the American college of Gastroenterology and its practice parameters committee provided a definition of Barrett's esophagus "as a change in the esophageal epithelium of any length that can be recognised at endoscopy and is confirmed to have intestinal metaplasia by biopsy."[25] Thus the presence of goblet cells has become the accepted diagnostic criterion of this disease, regardless of the precise site of biopsy within the tubular esophagus.

Barrett's esophagus (BE) is an acquired condition in which the squamous epithelium at the distal esophagus is replaced by metaplastic columnar epithelium. In general, BE results from severe esophageal mucosal injury by chronic gastroesophageal reflux disease[25,34,41,43,51]. Interest in BE mainly stems from its well established association with adenocarcinoma of the esophagus, a cancer whose incidence has increased rapidly in the last 3 decades. The recognition that cancer in BE predominantly originates from intestinal type columnar epithelium has led to the definition of BE by the presence of intestinal metaplasia (IM) in the esophagus[34]. The diagnosis of BE is readily achieved in patients who have long segments of IM by correlating endoscopic and histological findings.

To avoid over diagnosis of BE, its presence is considered to be established only if endoscopic examination reveals a displacement of the squamocolumnar junction and if IM is detected by histological examination[34].

Barrett's esophagus is considered to be a disorder of the Caucasian male and therefore presumed to be rare among non-Caucasian populations. However, recent reports indicate that its occurrence among populations of the developing countries might have been underestimated. Gadour and Ayoola study indicates that 0.3% of all patients undergoing endoscopy for various indications have BE. In their study 5 of 8 cases of BE were males. The number was too small for any firm conclusions regarding the sex distribution[17].

It is clearly established that both the intestinal metaplasia associated with Barrett's esophagus and its subsequent progression to esophageal adenocarcinoma are associated with pathologic gastro esophageal reflux.

Gastroesophageal reflux associated with the development of intestinal metaplasia is generally both severe and long standing, and is characterised by profound alterations in the esophageal luminal environment, including frequent and wide fluctuations in luminal PH

and high exposure to inflammatory components such as bile salts and proteolytic enzymes.

A large body of research over the past decade has outlined many of the genetic changes that are important in the metaplastic-dysplastic-carcinoma progression of esophageal adenocarcinoma. Implicated genes, including those involved in cell signaling, cell cycle control, cell adhesion, and apoptosis are well characterised and their expression levels are known to be altered in esophageal carcinogenesis.

Endoscopic surveillance of patients with Barrett's esophagus is recommended to detect malignancy at an early, potentially curable stage. However, it has been argued that surveillance is relatively ineffective and not very cost effective. This has led to a search for risk factors that allows for better stratification of the patients according to their individual risk of developing adencarcinoma.

Not only the history of esophageal carcinoma but also risk factors for developing of such cancer could differ over time and from one region to another. Squamous cell carcinoma of the esophagus in the developed world is at least five times more common in men than in women, properly because men drink and smoke more[37].

A diversity of begnin tumors and nonneoplastic masses can be seen in the esophagus. They are however mostly uncommon lesions, small and asymptomatic, whose importance lies in their distinction from malignant tumors. Most clinically apparent tumors grow or protrude into the lumen and thus appears as polyps at endoscopy. The differential

diagnosis of polypoid esophageal lesions[25] is summarised in following Table No X.

Differential diagnosis of polypoid esophageal Tumors

Table No X

LESION	DISTINGUISHING FEATURES
Benign Squamous Papilloma	Bland epithelium covering fibrovascular cores
Fibrovascular Polyp	Pedunculated mass of fibrous (+/- adipose) tissue found in upper esophagus.
Inflammatory fibroid polyp	Vascular fibroblastic tissue with mixed inflammation
Submucosal tumors	Leiomyoma, granular cell tumor most common
Malignant Squamous cell carcinoma	Especially verrucous variant
Adenocarcinoma	Infrequently polypoid
Sarcomatoid carcinoma	Atypical spindle cell stroma predominates
Malignant melanoma	Melanin pigmentation; junctional changes
Sarcomas	Rare, distinguish from carcinoma with spindle cell component
Metastic tumors	Occasionally produce polypoid mass

In our study 81 cases of noncancerous lesion is observed in which 42 cases are with squamous intra epithelial neoplasm (SIL) changes and 14 cases show only normal esophageal epithelium and 14 cases with only fibrous tissue without the covering epithelium.

1 case of squamous papilloma showing benign squamous epithelium lining delicate connective tissue stalk is seen. Squamous papillomas are the most common benign epithelial esophageal neoplasm mostly arising in the distal esophagus appearing as small and sessile mass, usually solitary.

They are rare lesion, noted in less than 0.04% of endoscopic examination and typically represent an incidental finding in asymptomatic patients, although larger lesions cause dysphagia. Several studies have identified HPV DNA in some squamous papilloma particularly HPV 6,11,16,18.[44]

One case of fibromuscular polyp with intact overlying mucosa and core of mature fibrous tissue exhibiting foci of myxoid areas, and thin walled vessels is seen.

Typically these polyps arise in the upper esophagus from the cricopharyngeal region and they present as elongated pedunculated mass that may range upto 20 cm in length.

Squamous cell carcinoma is the most common malignant tumor of esophagus worldwide. It affects predominantly men with peak incidence in 5th decade of life. Its incidence differs dramatically, however, in different countries with smoking, alcohol abuse, dietary exposure, genetic factors and HPV all suspected of playing etiological role. Esophageal cancer poses several general diagnostic task for surgical pathologist;

Establish the diagnosis of malignancy, classifying the histological type and assessing prognostic factor.

The recognition of esophageal malignancy in mucosal biopsy specimen is usually straightforward although the diagnostic features may be hidden by more abundant, inflamed, fibrotic or necrotic tissue. Cytological examination is an effective valuable adjunct. Diagnostic yield of combined endoscopic biopsy and brush cytology approaches 100%[42].

In our study 176 cases of squamous cell carcinoma and 18 cases of Adenocarcinoma is initially diagnosed with endoscopic biopsy evaluation and the ratio between squamous cell carcinoma and adenocarcinoma is 8.2:1.

This figures come in correlation with studies conducted by various author in the developing countries especially in south Asian countries. But in contrast the studies conducted by western people show an increased incidence of adenocarcinoma almost with 1:1 ratio, probably attributed to their changes in lifestyle, increased intake of alcohol, smoking, food habits etc. But our study correlates well with both south Asians as well as western research workers that both in SCC and Adenocarcinoma, the incidence is more common in male.

Squamous cell carcinoma are usually located in the lower and midesophagus, only about 10% are found in cervical and upper thoracic region[32,51]. In accordance, in our study only 4.35% are found in cervical and 10.86% are found in middle and 73.91% are found in lower esophagus.

At the time of diagnosis, most are advanced lesions with invasion into or beyond the muscularis propria. In our study only one esophagectomy specimen revealed adjacent nodal metastasis. Grossly SCC usually present as fungating, proliferative mass[51] , in our study also 39 cases (84.78%) presented as fungating proliferative mass .

Microscopically SCC are classified into well, moderate and poorly differentiated and any degree of differentiation may occur and variation within single tumour is common. In better differentiated lesions, mild nuclear atypia and cellular pleomorphism are accompanied by readily identified keratinisation, whereas in poorly differentiated tumors, keratinisation is sparse and cytological abnormalities more pronounced.

One case of sarcomatoid carcinoma with biphasic histology exhibiting both carcinomatous and spindle cell components, on Immunohistochemistry revealed features of sarcoma with vimentin positivity.

The assortment of names applied to this Tumor- Carcinosarcoma, pseudosarcoma, pseudosarcomatous squamous cell carcinoma, spindle cell carcinoma and polypoid carcinoma testifies to differing views of its histogenesis and biology.

The predominant concept is that it originates by sarcomatous metaplasia of malignant epithelial cells.

One case of basaloid squamous cell carcinoma with characteristic invasive lobules of basaloid cells with peripheral palisading, areas of necrosis and deposits of hyalinized basal lamina stroma, which tends to be deeply invasive, with frequent widespread metastases which is of poor prognosis was seen.

One case of adenosquamous carcinoma characterised by mixed glandular and squamous differentiation in more equal proportions is noted which is a rare aggressive neoplasm. One case of primary malignant melanoma characterised by basal proliferation of benign melanocytes (Melanosis) with the stroma infiltrated by pigmented epithelioid and spindled cells in sheets was seen, which on Immunohistochemistry showed positivity for S100.

Most adenocarcinomas develop in the lower third of esophagus, some may extend into the proximal stomach. Grosss appearance being flat irregular plaques to advanced fungating masses. Most are well or moderately differentiated. Intestinal type glandular formulation is common.

The intestinal metaplastic epithelium of Barrett’s esophagus can be identified adjacent to the tumor in most cases, and epithelial dysplasia is a common coexisting feature. In some tumors however, no residual Barrett’s esophagus is seen, presumably because it has been overgrown by the carcinoma.

CONCLUSION

The present study comprising of 277 esophageal biopsies and 46 esophagectomy specimens suggest the following conclusions.

1. The incidence of esophageal lesions is 9.75%
2. Esophageal lesions are common in males, with Male: Female ratio of 2.2: 1
3. Esophageal lesions both neoplastic and non neoplastic are common in the lower end of esophagus
4. The habit of tobacco chewing along with betel nuts and leaves and consuming alcohol is considered to be major predisposing factor in our region
5. Nonneoplastic esophageal lesions are more prevalent in 31-40 years of age group.
6. Neoplastic esophageal lesions are more prevalent in 51-60 yrs of age group.
7. Most common presenting symptom is dysphagia observed in all the patients under study, followed by loss of weight and anorexia.
8. Most of the cancers are of squamous cell origin.
9. Barret's esophagus is noted in 3.25% of patients with symptomatic GERD who undergo endoscopy with biopsy.
10. Barret's esophagus with dysplasia progress to adenocarcinoma.
11. Immunohistochemistry remains in its place as final confirmative tool in doubtful cases.

APPENDIX I

Procedure of combined AB PH 2.5 PAS FOR ACID AND NEUTRAL MUCINS

Acid mucins and neutral mucins are clearly separated by this technique. The rationale is that by first staining all acid mucins with Alcian Blue, those acid mucins which are also PAS positive will not react with the subsequent PAS reaction, neutral mucins alone will take up PAS stain. In this way a good colour distinction can be made between acid and neutral mucins.

Preparation of Stains:

a) Alcain Blue-1gm 3% Acetic acid –100cm^3

b) Schiff's Reagent- 1gm basic fuschin in 200cc boiling water

   i. 2gm Potassium metabisulfite

   ii. 2ml Concentrated HCL

   iii. Mix and add 2gm activated charcoal –filter and store at 4°C

c) 1% aqueous periodic acid.

METHOD:

1. Dewax sections and bring to water
2. Alcian Blue solution - 5 minutes
3. Wash in distilled water
4. 1% aqueous periodic acid - 5 minutes
5. Rinse well in distilled water
6. Schiff's reagent - 15 minutes
7. Wash in running tap water - 5-10 minutes
8. Stain nuclei lightly with Harris Haematoxylin
9. Differentiate as appropriate and blue.
10. Wash in distilled water
11. Rinse in absolute alcohol
12. Clean in Xylene and mount as desired

Results:

Acid mucin – Blue

Neutral mucin – Magenta

Mixtures of the above – the colour will develop on the dominant entity and will range from blue purple, through purple to a violet or magenta colour.

APPENDIX – II

STANDARD HEMATOXYLIN AND EOSIN STAIN FOR PARAFFIN SECTIONS

Method:

1. De wax sections, hydrate through graded alcohols to water.
2. Remove fixation pigments.
3. Stain in an alum hematoxylin of choice for a suitable time.
4. Wash well in running tap water until sections ‘blue’ for 5 minutes or less.
5. Differentiate in 1 percent acid alcohol (1 percent HCL in 70 percent alcohol) for 5-10 sec.
6. Wash well in tap water until sections are again ‘blue’ (10-15) minutes), or
7. Blue by dipping in a alkaline solution (e.g. ammonia water), followed by a 5-min. tap water wash.
8. Stain in 1 percent eosin Y for 10 min.
9. Wash in running tap water for 1-5 min.
10. Dehydrate through alcohols, clear and mount.

Results:

Nuclei	blue/black
Cytoplasm	Varying shades of pink
Muscle fibers	deep pink/red
Red blood cells	orange/red
Fibrin	deep pink

Notes:

Note that structures and substances other than nuclei may be hematoxphilic to varying degrees. Examples include fungal hyphae, which are faintly hematoxyphilic, and calcium deposits, which are often deep blue- black.

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S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
1	4/2000	60	M	620660	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Squamous cell carcinoma
2	32/2000	40	M	3415/99	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Well differentiated keratinising squamous cell carcinoma
3	34/2000	35	F	621017	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Well differentiated keratinising squamous cell carcinoma
4	35/2000	80	F	620151	Dysphagia, Anorexia, Dyspepsia, Vomiting	Esophagitis	Focal SIL changes
5	118/2000	25	F	622838	Dysphagia, Loss of Weight, Anorexia	Growth 28 cm down	Well differentiated keratinising squamous cell carcinoma
6	119/2000	70	F	623044	Dysphagia, Loss of Weight, Anorexia	Growth 28 cm down	Well differentiated squamous cell carcinoma
7	127/2000	37	F	623475	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Well differentiated squamous cell carcinoma
8	161/2000	50	M	623925	Odynophagia, Dyspepsia, vomiting	Barrett's esophagus	Barrett's esophagus
9	162/2000	50	M	703120	Odynophagia, Dyspepsia, vomiting	Growth esophagus	Squamous papilloma
10	175/2000	50	F	624303	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Squamous cell carcinoma
11	176/2000	50	F	624400	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Squamous cell carcinoma
12	386/2000	38	M	634014	Dysphagia, Dyspepsia, vomiting	Oesophaual nodule	SIL changes
13	236/2000	45	M	703952	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Moderately differentiated keratinising SCC
14	242/2000	50	M	625779	Dysphagia, Dyspepsia, vomiting	OG junction growth	Focal SIL changes
15	251/2000	30	F	625706	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Well differentiated squamous cell carcinoma
16	257/2000	40	F	625767	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Well differentiated squamous cell carcinoma
17	288/2000	30	F	626684	Dysphagia, Loss of Weight, Anorexia	Esophageal growth	Invasive SCC
18	290/2000	25	F	626654	Dysphagia, Loss of Weight, Anorexia	Growth lower end of esophagus	Well differentiated squamous cell carcinoma
19	319/2000	64	F	627315	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Squamous cell carcinoma
20	364/2000	47	F	628334	Dysphagia, Loss of Weight, Anorexia	Carcinoma Mid esophagus	Invasive SCC
21	388/2000	65	F	629134	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Early SCC
22	391/2000	40	M	709375	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Well differentiated squamous cell carcinoma
23	489/2000	60	M	632202	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Barrett's with adenocarcinoma
24	528/2000	45	M	631904	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	keratinising squamous cell carinoma
25	531/2000	50	F	680606	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus lower end	Moderately differentiated SCC
26	541/2000	35	F	632526	Dysphagia, Loss of Weight, Anorexia	Growth 23 to 30 cm	Well differentiated squamous cell carcinoma
27	542/2000	70	M	630709	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus 20 cm down	Well differentiated infiltrating SCC
28	603/2000	60	M	633601	Dysphagia, Loss of Weight, Anorexia	Growth	Well differentiated infiltrating SCC
29	704/2000	65	M	635639	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Squamous cell carcinoma
30	814/2000	50	M	637316	Dysphagia, Loss of Weight, Anorexia	Lower end growth	Well differentiated infiltrating SCC
31	836/2000	42	M	638267	Dysphagia, Loss of Weight, Anorexia	Growth 20 cm down	Non keratinising invasive SCC
32	863/2000	50	M	638198	Dysphagia, Loss of Weight, Anorexia	Growth 35 cm	Well differentiated invasive SCC
33	889/2000	50	F	638963	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Well differntiated SCC
34	948/2000	50	M	640315	Dysphagia, Loss of Weight, Anorexia	Oesophagus growth	Well differntiated SCC

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
35	967/2000	50	F	640236	Dysphagia, Anorexia, Dyspepsia, Vomiting	OG junction growth	SIL changes
36	989/2000	35	M	640569	Dysphagia, Loss of Weight, Anorexia	Esophagus growth at 25 cm	Well differentiated infiltrating SCC
37	1013/2000	60	M	640955	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Moderately differentiated SCC
38	1074/2000	50	F	641851	Dysphagia, Anorexia, Dyspepsia, Vomiting	OG junction growth	SIL changes
39	1216/2000	55	M	643186	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Moderately differentiated SCC
40	1253/2000	50	M	642892	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Infiltrating Adenocarcinoma
41	1295/2000	50	M	1902/2000	Dysphagia, Anorexia, Dyspepsia, Vomiting	Growth 34 cm down	Focal SIL changes
42	1296/2000	80	M	2103/2000	Dysphagia, Loss of Weight, Anorexia	Growth 34 - 37 cm down	Well differentiated infiltrating SCC
43	1319/2000	65	M	645202	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Moderately differentiated SCC
44	1344/2000	45	F	645112	Dysphagia, Loss of Weight, Odynophagia	OG junction growth	Moderately differentiated infiltrating SCC
45	1400/2000	50	F	647282	Dysphagia, Loss of Weight, Odynophagia	Oesophagus growth	Poorly differentiated SCC
46	1448/2000	50	M	647791	Dysphagia, Odynophagia, Dyspepsia, Vomiting	Growth esophagus	SIL changes
47	1458/2000	60	M	647951	Dysphagia, Loss of Weight, Odynophagia	Ulcer esophagus	Moderately differentiated infiltrating SCC
48	1465/2000	60	M	647111	Dysphagia, Loss of Weight, Odynophagia	Growth esophagus	Well differentiated SCC
49	1466/2000	80	F	648423	Dysphagia, Loss of Weight, Odynophagia	OG junction growth	Moderately differentiated SCC
50	401/2001	53	M	669670	Dysphagia, Loss of Weight, Odynophagia	OG junction growth	Squamous cell carcinoma
51	468/2001	63	M	671256	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Growth esophagus	Infiltrating SCC
52	490/2001	78	M	668913	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
53	491/2001	30	M	668840	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
54	492/2001	23	M	668915	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
55	493/2001	50	M	670565	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
56	494/2001	35	M	670999	Dysphagia, Loss of Weight, Odynophagia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
57	495/2001	25	M	671078	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
58	496/2001	26	M	671545	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
59	564/2001	65	M	663059	Dysphagia, Loss of Weight, Anorexia	30 cm esophagus	Moderately differentiated SCC
60	565/2001	50	M	673048	Dysphagia, Anorexia, Dyspepsia, Vomiting	Growth 18 cm	No tissue
61	645/2001	50	M	674013	Dysphagia, Loss of Weight, Anorexia	Growth OG junction	Well differentiated SCC
62	660/2001	60	F	674861	Dysphagia, Loss of Weight, Anorexia	Growth 20 - 25 cm	Well differentiated infiltrating SCC
63	668/2001	65	M	674578	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Moderately differentiated SCC
64	714/2001	60	M	675698	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Well differentiated SCC
65	761/2001	25	M	676090	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Well differentiated SCC
66	787/2001	66	M	677178	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Moderately differentiated SCC
67	817/2001	30	F	676332	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
68	818/2001	29	M	676435	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
69	819/2001	60	M	675950	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Normal stratified squamous epithelium
70	820/2001	58	M	676100	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Focal SIL changes
71	821/2001	60	F	676472	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Normal histology
72	892/2001	67	M	98039	Dysphagia, Anorexia, Dyspepsia, Vomiting	Growth esophagus	SIL changes
73	893/2001	45	F	67898	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Well differentiated SCC
74	1016/2001	56	M	680973	Dysphagia, Loss of Weight, Anorexia	Growth esophagus 30 cm	Non keratinising SCC
75	1119/2001	85	M	777576	Dysphagia, Loss of Weight, Anorexia	Growth middle 3rd esophagus	Moderately differentiated infiltrating SCC
76	1181/2001	60	M	774728	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus	Moderately differentiated infiltrating SCC
77	1223/2001	45	F	684678	Dysphagia, Loss of Weight, Anorexia	Growth 30 cm	Moderately differentiated SCC
78	1460/2001	50	F	688376	Dysphagia, Loss of Weight, Anorexia	Growth 20 cm down	keratinising infiltrating SCC
79	1496/2001	28	M	689158	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Hyperplastic stratified squamous epithelium with focal lowgrade SIL changes
80	1498/2001	45	M	689666	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Well differentiated SCC
81	1499/2001	65	M	688978	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus 20 cm	Well differentiated infiltrating SCC
82	1516/2001	70	M	689595	Dysphagia, Loss of Weight, Anorexia	Growth esophagus	Well differentiated infiltrating SCC
83	1517/2001	67	M	689551	Dysphagia, Loss of Weight, Anorexia	Carcinoma esophagus 35 cm down	Well differentiated Adenocarcinoma
84	1533/2001	55	M	689772	Dysphagia, Loss of Weight, Anorexia	OG junction growth	Well differentiated infiltrating SCC
85	1549/2001	80	M	690033	Dysphagia, Loss of Weight, Anorexia	Esophageal growth	Necrotic tissue

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
86	1631/2001	49	F	691489	Dysphagia, Loss of Weight, Heart burn	Growth esophagus 30 cm down	keratinising infiltrating SCC
87	1641/2001	69	M	691846	Dysphagia, Loss of Weight, Heart burn	Carcinoma esophagus 28 cm	Non keratinising SCC
88	1737/2001	30	M	692101	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
89	1738/2001	35	F	692459	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
90	1740/2001	45	F	688626	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
91	1741/2001	38	M	692568	Dysphagia, Anorexia, Dyspepsia, Vomiting	Barrett's esophagus	Barrett's esophagus
92	1783/2001	65	M	694124	Dysphagia, Loss of Weight, Anorexia, Heart burn	Esophageal growth	Well differentiated infiltrating non keratinising SCC
93	1805/2001	50	F	694469	Dysphagia, Loss of Weight, Anorexia, Heart burn	Carcinoma esophagus	Well differentiated infiltrating SCC
94	1812/2001	63	M	694108	Dysphagia, Loss of Weight, Anorexia, Heart burn	Carcinoma esophagus 20 cm	Well differentiated infiltrating SCC
95	1819/2001	35	F	694445	Dysphagia, Loss of Weight, Anorexia, Heart burn	30 cm down biopsy	Well differentiated infiltrating non keratinising SCC
96	1830/2001	47	M	693696	Dysphagia, Loss of Weight, Anorexia, Heart burn	Esophagus growth	Well differentiated infiltrating SCC
97	1936/2001	32	F	696463	Dysphagia, Loss of Weight, Anorexia, Heart burn	Esophagus growth	Well differentiated infiltrating SCC
98	1975/2001	78	M	697107	Dysphagia, Anorexia, Dyspepsia, Vomiting	Growth esophagus	SIL changes
99	2073/2001	60	M	699593	Dysphagia, Loss of weight, Anoroxia, Heart burn	Growth esophagus 20 cm down	Moderately differentiated infiltrating SCC
100	2116/2001	58	M	700760	Dysphagia, Loss of weight, Anoroxia, Heart burn	Carcinoma esophagus	Well differentiated infiltrating SCC
101	2246/2001	60	M	700956	Dysphagia, Loss of weight, Anoroxia, Heart burn	Growth esophagus	Well differentiated keratinising SCC
102	2272/2001	32	M	703081	Dysphagia, Loss of weight, Anoroxia, Heart burn	25 cm growth	Well differentiated keratinising SCC
103	2297/2001	38	M	702910	Dysphagia, Loss of weight, Anoroxia, Heart burn	Growth esophagus	Well differentiated keratinising SCC
104	1248/2001	40	F	684763	Dysphagia, Loss of weight, Anoroxia, Heart burn	Growth esophagus	Well differentiated keratinising SCC
105	1319/2001	65	M	685882	Dysphagia, Loss of weight, Odynophagia, Heart burn	Growth esophagus lower end	Well differentiated keratinising SCC

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
106	1417/2001	78	M	688371	Dysphagia, Loss of weight, Odynophagia, Heart burn	OG junction growth	Well differentiated Adenocarcinoma
107	1418/2001	58	F	688360	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Carcinoma esophagus 30 cm	High grade SIL
108	1429/2001	50	F	688631	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Growth 20 - 28 cm	SIL changes
109	75/2002	40	M	704127	Dysphagia, Loss of Weight, Odynophagia	Growth esophagus	Moderately differentiated infiltrating SCC
110	104/2001	55	M	705486	Dysphagia, Loss of Weight, Odynophagia	Growth lower end of esophagus	Well differentiated Adenocarcinoma
111	157/2002	30	F	704820	Anaemia, Dysphagia, Loss of Weight	Ulcerative esophagus	Well diferentiated nonkeratinising SCC
112	159/2002	58	M	706116	Dysphagia, Loss of weight, Anorexia, Hoarseness of voice	Esophagus growth	High grade infiltrating SCC
113	170/2002	70	M	706628	Dysphagia, Loss of weight, Odynophagia, Heart burn	Growth mid esophagus	Well diferentiated SCC
114	226/2002	45	F	707194	Dysphagia, Loss of weight, Odynophagia, Heart burn	Esophageal growth biopsy	Well differentiated Keratinising infiltrating SCC
115	271/2002	40	F	707738	Dysphagia, Loss of weight, Odynophagia, Heart burn	Growth middle 1/3rd	Well differentiated SCC
116	303/2002	52	M	708511	Dysphagia, Loss of weight, Odynophagia, Heart burn	Esophageal growth biopsy	Intra epithelial carcinoma
117	304/2002	50	M	708253	Dysphagia, Loss of weight, Odynophagia, Heart burn	Esophageal growth biopsy	Mucin secreting adenocarcinoma
118	426/2002	62	F	710438	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Esophageal growth biopsy	SIL changes
119	496/2002	58	M	711872	Dysphagia, Loss of weight, Odynophagia, Heart burn	Esophageal growth 25 cm	Moderately differentiated SCC
120	660/2002	70	M	814051	Dysphagia, Loss of weight, Odynophagia, Heart burn	Carcinoma esophagus lower 1/3rd	No esophageal mucosa
121	869/2002	43	M	718245	Dysphagia, Loss of weight, Odynophagia, Heart burn	Carcinoma esophagus	Moderately differentiated SCC
122	890/2002	40	F	717761	Dysphagia, Loss of weight, Odynophagia, Heart burn	Growth 30 cm down	Well differentiated SCC
123	904/2002	55	M	710394	Dysphagia, Loss of weight, Odynophagia, Heart burn	OG junction growth	Early Squamous cell carcinoma with Barrett's esophagus
124	905/2002	50	F	718770	Dysphagia, Loss of weight, Odynophagia, Heart burn	OG junction growth	Moderately differentiated SCC
125	966/2002	45	M	718772	Dysphagia, Loss of weight, Odynophagia, Heart burn	OG junction growth	Moderately differentiated adenocarcinoma
126	1038/2002	40	F	720445	Dysphagia, Loss of weight, Odynophagia, Heart burn	Oesophagial growth Biopsy	Invasive SCC

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127	1166/2002	50	F	722181	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Oesophagial growth Biopsy	Low grade SIL
128	1295/2002	50	M	725387	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Oesophagial growth Biopsy	High grade SIL
129	1319/2002	48	M	725092	Dysphagia, Loss of Weight, Anorexia, Heart burn	Oesophagial growth Biopsy	Moderately differentiated SCC
130	1401/2002	57	M	727152	Dysphagia, Loss of Weight, Anorexia, Heart burn	Growth upper esophagus	Moderately differentiated SCC
131	1402/2002	75	M	726122	Dysphagia, Loss of Weight, Anorexia, Heart burn	Mid esophagus, ulcer	Well differentiated SCC
132	1434/2002	50	M	2143/02	Dysphagia, Loss of Weight, Anorexia, Heart burn	Growth esophagus	Moderately differentiated SCC
133	1476/2002	75	F	728004	Dysphagia, Loss of weight, Anorexia	Mid esophagus, proliferative growth	Moderately differentiated SCC
134	1512/2002	62	M	728147	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	OG junction growth	High grade SIL
135	1592/2002	70	F	729501	Dysphagia, Loss of Weight, Anorexia, Heart burn	OG junction growth	Moderately differentiated SCC
136	1616/2002	55	F	729699	Dysphagia, Loss of Weight, Anorexia, Heart burn	OG junction growth	Invasive SCC
137	1635/2002	49	F	831557	Dysphagia, Loss of Weight, Anorexia, Heart burn	Osophageal growth	Moderately differentiated SCC
138	1701/2002	55	M	730468	Dysphagia, Loss of Weight, Anorexia, Heart burn	Osophageal growth	Invasive SCC
139	1702/2002	40	F	730460	Dysphagia, Loss of Weight, Anorexia, Heart burn	Osophageal growth	Invasive SCC
140	1705/2002	45	M	833275	Dysphagia, Loss of Weight, Anorexia, Heart burn	OG junction growth	Moderately differentiated SCC
141	1727/2002	40	M	730981	Dysphagia, Loss of weight, Odynophagia, Heart burn	Esophageal growth	Moderately differentiated SCC
142	1768/2002	55	F	731868	Dysphagia, Loss of weight, Odynophagia	Esophageal growth	Moderately differentiated SCC
143	1834/2002	50	F	732500	Dysphagia, Odynophagia, Dyspepsia, Vomiting	Mid esophageal ulcer	Low grade SIL
144	1854/2002	60	M	733387	Dysphagia, Loss of weight, Odynophagia	Growth esophagus	Moderately differentiated Adenocarincoma
145	1856/2002	50	F	73378	Dysphagia, Loss of weight, Odynophagia	Growth esophagus 30 cm down	Moderately diferentiated infiltrating SCC
146	1882/2002	50	M	733392	Dysphagia, Loss of weight, Odynophagia, Heart burn	Growth 33 - 35 cm	Moderately diferentiated infiltrating SCC

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147	1885/2002	50	M	733878	Dysphagia, Loss of Weight, Odynophagia	Growth esophagus	Well differentiated SCC
148	1915/2002	47	M	733972	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	OG junction growth	Focal low grade SIL
149	1950/2002	50	M	734619	Dysphagia, Loss of weight, Anorexia	Growth upper esophagus	Moderately differentiated SCC
150	2017/2002	70	M	735227	Dysphagia, Loss of Weight, Anorexia, Heart burn	Upper esophageal growth	Well differentiated invasive SCC
151	2044/2002	45	M	735269	Dysphagia, Loss of weight, Anorexia	Growth 35 cm	Moderately differentiated SCC
152	2094/2002	50	M	736326	Dysphagia, Loss of weight, Anorexia	Esophageal growth biopsy	Moderately differentiated SCC
153	2095/2002	50	F	734552	Dysphagia, Loss of Weight, Anorexia, Heart burn	OG junction growth	Moderately differentiated SCC
154	2159/2002	50	M	737582	Dysphagia, Loss of weight, Anorexia	Growth 30 cm down obstructure	Moderately differentiated SCC
155	2182/2002	45	M	735269	Dysphagia, Loss of Weight, Anorexia, Heart burn	Growth 30 cm down	Moderately differentiated SCC
156	2187/2002	65	M	842514	Dysphagia, Loss of weight, Anorexia	OG junction growth	Moderately differentiated adenocarcinoma
157	2190/2002	60	M	841061	Dysphagia, Loss of weight, Anorexia	Carcinoma esophagus	Moderately differentiated SCC
158	2240/2002	60	M	738562	Dysphagia, Anorexia, Dyspepsia, Vomiting	Carcinoma esophagus	No tissue
159	2250/2002	65	M	639042	Dysphagia, Loss of weight, Anorexia	Growth firm 33 cm	Moderately differentiated adenocarcinoma
160	2273/2002	65	M	739643	Dysphagia, Anorexia, Dyspepsia, Vomiting	Growth esophagus	No tissue
161	2391/2002	60	M	738562	Dysphagia, Loss of Weight, Anorexia, Heart burn	Growth esophagus	Moderately differentiated SCC
162	2476/2002	65	F	742303	Dysphagia, Loss of weight, Anorexia	Growth esophagus	Moderately differentiated SCC
163	2494/2002	55	F	848087	Dysphagia, Loss of Weight, Anorexia, Heart burn	Growth esophagus	Well differentiated SCC
164	2570/2002	76	F	849356	Dysphagia, Loss of weight, Anorexia	Growth esophagus	Well differentiated SCC
165	2/2003	55	M	744709	Dysphagia, Loss of Weight, Anorexia, Heart burn	Carcinoma Esophagus	Moderately differentiated non keratinising SCC
166	5/2003	60	M	744717	Dysphagia, Loss of weight, Anorexia	Carcinoma Esophagus	Adenocarcinoma
167	43/2003	55	F	744647	Dysphagia, Loss of weight, Anorexia	Carcinoma esophagus lower 1/3rd	Moderately differentiated SCC
168	46/2003	43	M	744783	Dysphagia, Loss of weight, Anorexia	Carcinoma Esophagus	Moderately diferentiated infiltration SCC
169	47/2003		F	745107	Dysphagia, Odynophagia, Heart burn	Carcinoma Esophagus	Low grade SIL
170	64/2003		M	745089	Dysphagia, Loss of Weight, Heart burn	Middle 1/3rd	Infiltrating SCC
171	76/2003	60	M	850304	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Carcinoma Esophagus	No tissue
172	127/2003	65	M	746798	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	OG junction growth	SIL

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
173	186/2003	50	F	785300	Dysphagia, Loss of weight, Regurgitation of food, Heart burn	Dysphagia for Evaluation	Moderately differentiated SCC
174	308/2003	50	M	860998	Dysphagia, Loss of weight, Regurgitation of food, Dyspepsia, Vomiting	Carcinoma Esophagus	Moderately differentiated SCC
175	642/2003	52	M	866569	Dysphagia, Odynophagia, Regurgitation of food, Heart burn	Carcinoma Esophagus	No tissue
176	693/2003	68	M	755772	Dysphagia, Loss of weight, Regurgitation of food, Heart burn	Carcinoma OG Junction	Non keratinising SCC
177	694/2003	35	F	868125	Dysphagia, Loss of weight, Regurgitation of food, Heart burn	Mid esophagus growth	Moderately differentiated infiltrating SCC
178	730/2003	45	F	868125	Dysphagia, Anorexia, Dyspepsia, Vomiting	Mid esophagus growth	No tissue
179	731/2003	45	M	868543	Dysphagia, Anorexia, Odynophagia, Dyspepsia, Vomiting	Carcinoma Esophagus with Submucous fibrosis	Normal stratified squamous epithelium
180	763/2003	60	M	756818	Dysphagia, Loss of weight, Regurgitation of food	Mid esophagus growth	Moderately differentiated infiltrating SCC
181	825/2003		F	758555	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	growth esophagus	No tissue
182	832/2003	55	M	758565	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Mid esophagus growth	SIL
183	844/2003	47	M	759195	Dysphagia, Loss of weight, Regurgitation of food	Growth 30 cm down	Moderately differentiated adenocarcinoma
184	845/2003	52	M	759315	Dysphagia, Loss of weight, Regurgitation of food	Growth 25 cm down on dosc	Infiltrating SCC
185	846/2003	65	M	759256	Dysphagia, Loss of weight, Regurgitation of food	Growth Esophagus 30 cm down	Infiltrating SCC
186	856/2003	45	M	759400	Dysphagia, Loss of weight, Regurgitation of food	Growth Esophagus 30 cm down	Well differentiated SCC
187	858/2003	35	F	759736	Dysphagia, Loss of weight, Regurgitation of food	Growth Esophagus 30 cm down	Moderately differentiated SCC
188	887/2003	55	M	758500	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Lower esophagus growth	High grade SIL
189	970/2003	62	F	761784	Dysphagia, Loss of weight, Regurgitation of food	Esophagus Growth	Moderately differentiated keratinising SCC
190	994/2003	59	M	761707	Dysphagia, Anorexia, Regurgitation of food	Mid esophagus growth	Infiltrating Keratinising SCC
191	1037/2003	67	M	761919	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	OG junction growth	SIL changes
192	1038/2003	65	M	761980	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	OG junction	SIL changes

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
193	1050/2003	32	F	762131	Loss of weight, Anorexia, Regurgitation of food	Mid escphageal growth	Well differentiated SCC
194	1052/2003	55	M	763101	Anorexia, Odynophagia, Regurgitation of food, Dyspepsia, Vomiting	OG junction growth	No tissue
195	1130/2003	50	M	763403	Loss of weight, Anorexia, Odynophagia	Esophagus Growth OG junction	Well differentiated infiltrating SCC
196	S1196/2003	74	M	764101	Loss of weight, Anorexia, Odynophagia	Esophagus Growth OG junction	Mucoid carcinoma
197	1199/2003	60	M	765110	Dysphagia, Loss of weight, Anorexia	Carcinoma esophagus	Well differentiated SCC
198	1312/2003	55	M	766504	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Esophagus growth	SIL
199	1351/2003	60	F	763102	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Esophageal growth	High grade SIL
200	1376/2003	45	M	883704	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Growth upper 1/3rd esophagus	Low grade SIL
201	1490/2003	52	M	884300	Dysphagia, Loss of weight, Anorexia	Carcinoma esophagus	SCC with areas of H'age and necrosis
202	1508/2003		M	885767	Dysphagia, Loss of weight, Anorexia	Esophageal growth	Moderately differentiated SCC
203	1548/2003	40	M	886371	Dysphagia, Loss of weight, Anorexia	Growth Esophagus	Moderately differentiated SCC
204	1641/2003	55	M	772000	Dysphagia. Anorexia	Growth Esophagus	Normal statisfied squamous epithelium
205	1663/2003	55	M	773056	Dysphagia, Loss of weight, Regurgitation of food	Growth Esophagus	Well differentiated SCC
206	1697/2003	46	M	773672	Dysphagia, Anorexia, Regurgitation of food, Dyspepsia, Vomiting	Growth OG Junction	Hyperplastic squamous Epithelium with low grade SIL changes
207	1754/2003	61	F	456/03	Dysphagia, Odynophagia, Regurgitation of food	Oesophagial growth Biopsy	High grade SIL
208	1784/2003	46	M	773632	Dysphagia, Odynophagia, Regurgitation of food	OG junction growth	No tissue
209	1830/2003	35	M	774769	Dysphagia, Odynophagia, Regurgitation of food	OG junction growth	No tissue
210	1852/2003	65	M	775902	Dysphagia, Loss of weight, Odynophagia	Growth Esophagus	Moderately differentiated infiltrating SCC
211	1920/2003	54	M	775305	Dysphagia, Odynophagia, Regurgitation of food	? Carcinoma Esogphagus 25 cm	Mucosa with underlying granulation tissue
212	2146/2003	49	M	780927	Dysphagia, Loss of weight, Odynophagia	Carcinoma esophagus	Well diffrentiated infiltration SCC
213	2153/2003	30	M	781198	Dysphagia, Loss of weight, Regurgitation of food	Cardinoma esophagus	Infitrating SCC
214	2198/2003	55	F	782278	Dysphagia, Odynophagia, Regurgitation of food, Dypepsia, Vomiting	Cardinoma esophagus	Normal histology

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215	2294/2003	55	M	782997	Dysphagia, Loss of weight, Regurgitation of food	OG junction growth	Well differentiated SCC
216	2295/2003	50	M	78300	Dysphagia, Odynophagia, Regurgitation of food, Dypepsia, Vomiting	OG junction growth	SIL changes
217	8/2004	40	M	784461	Dysphagia, Loss of weight, Regurgitation of food	Carcinoma esophagus	Moderatly differentiated SCC
218	54/2004	50	F	784433	Odynophagia, Regurgitation of food	Carcinoma esophagus	Normal stratified squamous epithelium
219	76/2004	40	F	905394	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Moderatly differentiated keratinising SCC
220	137/2004	35	M	786461	Odynophagia, Regurgitation of food, Dypepsia, Vomiting	Growth Esophagus lower 1/3rd	No tissue
221	174/2004	50	M	786413	Odynophagia, Regurgitation of food, Dypepsia, Vomiting	Esophagus Growth	Low grade SIL
222	213/2004	35	M	786457	Odynophagia, Regurgitation of food, Dypepsia, Vomiting	Distal esophagus ulcer	No tissue
223	215/2004	55	M	788052	Loss of weight, Odynophagia, Regurgitation of food	OG junction growth	Moderately differentiated Papillary Adenocarcinoma
224	314/2004	65	M	788076	Loss of weight, Odynophagia, Regurgitation of food, Hoarseness of voice	Middle 1/3rd esophagus growth	Moderately differentiated infilltrating SCC
225	388/3004	66	M	780891	Dysphagia, Loss of weight, Regurgitation of food	OG Junction growth	Well differntiated infiltration SCC
226	447/2004	65	M	791232	Dysphagia, Loss of weight, Regurgitation of food	Mid esophageal growth	Moderately differentiated non keratinising SCC
227	536/2004	65	F	797300	Dysphagia, Loss of weight, Regurgitation of food	Ulcero proliferative growth at carciac end	Moderately differentiated non keratinising SCC
228	850/2004	59	M	798199	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
229	914/2004	55	M	798882	Dysphagia, Loss of weight, Regurgitation of food	? BARRETT'S esophagus	Adenocarcinoma
230	986/2004	37	M	800100	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
231	1021/2004	55	M	922699	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Well differentiated infiltration SCC
232	1118/2004	55	M	801625	Dysphagia, Loss of weight, Regurgitation of food	Lower 3rd esophagus	Well differentiated infiltration SCC
233	1192/2004	50	M	802911	Dysphagia, Loss of weight, Regurgitation of food	Ulcero proliferative growth at distal end	Moderately differentiated SCC
234	1300/2004	48	M	804697	Dysphagia, Loss of weight, Regurgitation of food	Growth OG junction	Moderately differentiated SCC with areas of necrosis

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235	1361/2004	55	F	930671	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated infiltrating SCC
236	1587/2004	70	M	930831	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Adenosquamous carcinoma
237	1590/2004	53	M	804848	Dysphagia, Regurgitation of food, Dypepsia, Vomiting	? Carcinoma Esogphagus	Stratified squamous epithelium with focal low grade SIL changes
238	1838/2004	40	M	810801	Dysphagia, Regurgitation of food	Growth esophagus	Normal statified squamous epithelium
239	1878/2004	62	M	810461	Dysphagia, Loss of weight	Growth esophagus	Moderately differentiated SCC
240	1906/2004	60	M	810770	Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated infiltrating SCC
241	1951/2004	60	M	813919	Loss of weight, Regurgitation of food	Growth OG junction	Well differentiated Adenocarcinoma
242	1979/2004	35	F	814390	Regurgitation of food, Dypepsia, Vomiting	Growth esophagus	High grade SIL with foci of infiltration
243	1980/2004	70	M	814399	Regurgitation of food, Dypepsia, Vomiting	Growth esophagus	SIL changes
244	1987/2004	50	M	942500	Regurgitation of food	Upper esophageal growth	Infiltrating Keratinising SCC
245	2178/2004	63	M	945579	Loss of weight, Regurgitation of food, Dypepsia, Vomiting	Carcinoma esophagus	Moderately differentiated infiltrating SCC
246	2294/2004	47	M	818304	Dysphagia, Regurgitation of food	Growth esophagus	SIL
247	2312/2004	47	M	818737	Dysphagia, Loss of weight	OG junction growth	Moderately differentiated SCC
248	2342/2004	60	M	819525	Dysphagia, Loss of weight	Middle 1/3rd esophagus growth	Moderately differentiated SCC
249	2448/2004	50	F	821147	Dysphagia, Loss of weight	Growth esophagus	Well differentitated SCC
250	2456/2004	47	F	821127	Dysphagia, Loss of weight	Growth esophagus	Moderately differentiated SCC
251	2487/2004	40	F	951901	Dysphagia, Loss of weight	Carcinoma esophagus	Well differentiated keratinising SCC
252	2776/2004	70	M	826357	Dysphagia, Regurgitation of food, Dypepsia, Vomiting	Carcinoma esophagus	SIL changes
253	2778/2004	50	M	827377	Regurgitation of food, Dypepsia, Vomiting	Carcinoma esophagus	SIL changes
254	54/2005	50	M	827370	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus at 22 cm	Well differentiated infiltrating SCC
255	70/2005	65	F	826465	Loss of weight, Odynophagia, Regurgitation of food	OG junction growth	Well differentiated adenocarcinoma
256	134/2005	64	M	859555	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
257	153/2005	56	M	818885	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
258	256/2005	60	F	819300	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Malignant melanoma
259	292/2005	60	M	831754	Loss of weight, Odynophagia, Regurgitation of food	OG junction growth	Moderately differentiated SCC

S. No.	Patho No.	Age	Sex	IP. No.	Symptoms	Endoscopic Picture	HPE
260	318/2005	55	M	831977	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Sarcormatoid carcinoma
261	340/2005	64	M	832092	Loss of weight, Odynophagia, Regurgitation of food	Carcinoma esophagus	Moderately differentiated SCC
262	461/2005	55	M	833532	Loss of weight, Odynophagia, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
263	460/2005	35	M	833830	Dysphagia, Odynophagia, Regurgitation of food, Dypepsia, Vomiting	Growth esophagus	SIL changes
264	462/2005	40	M	833844	Dysphagia, Odynophagia, Regurgitation of food	Growth OG junction	Normal stratified squamous epithelium
265	487/2005	50	M	964964	Dysphagia, Regurgitation of food	? Carcinoma esophagus	Stratified squamous cells with koilocytic changes no frank malignancy
266	575/2005	20	M	966302	Dysphagia, Loss of weight, Regurgitation of food	Growth lower 3rd of esophagus	High grade infilltrating SCC
267	640/2005	55	M	969605	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
268	644/2005	27	M	968092	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
269	814/2005	38	M	838868	Dysphagia, Regurgitation of food	Oesophageal polyp	Polyp
270	1037/2005	55	F	841660	Dysphagia, Loss of weight, Regurgitation of food	Growth middle 3rd esophagus	Moderately differentiated SCC
271	1182/2005	66	M	840216	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
272	1443/2005	55	M	980327	Dysphagia, Loss of weight, Regurgitation of food	Growth middle 3rd esophagus	Moderately differentiated SCC
273	1490/2005	68	M	981345	Dysphagia, Loss of weight, Regurgitation of food, Dyspepsia, Vomiting	? Scleroma of esophagus (growth)	SIL changes
274	1619/2005	50	M	847260	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus lower 3rd	Moderately differentiated SCC
275	1638/2005	48	F	849886	Dysphagia, Loss of weight, Regurgitation of food	Growth esophagus	Moderately differentiated SCC
276	1696/2005	66	F	850011	Dysphagia, Loss of weight, Regurgitation of food	Esophageal growth	Moderately differentiated SCC
277	2278/2005	55	F	850012	Dysphagia, Loss of weight, Regurgitation of food	Esophageal growth	Well differentiated Adenocarcinoma

S. No.	Patho No.	Age	Sex	IP No.	Site of growth	Type of growth	HPE
1	139/2000	35	F	621017	Lower 1/3rd	Fungating	Well differentiated SCC
2	1162/2000	52	M	642463	Lower 1/3rd	Fungating	Well differentiated SCC
3	226/2001	65	F	623634	Middle 1/3rd	Fungating	Moderately differentiated SCC
4	871/2001	66	M	677178	Lower 1/3rd	Ulcerative	High grade SCC with areas of basi squamoid picture
5	1209/2001	53	M	680793	Lower 1/3rd	Fungating	Moderately differentiated SCC
6	1218/2001	28	M	68246	Lower end	Ulcerative	Moderately differentiated Adenocarcinoma
7	1459/2001	55	F	688173	Lower end	Fungating	Moderately differentiated SCC
8	1656/2001	45	M	689666	Lower end	Fungating	Well differentiated SCC
9	2145/2001	47	F	700372	Lower end	Fungating	Basaloid squamous cell carcinoma
10	1448/2002	45	F	720445	OG junction	Fungating	Well differentiated SCC
11	1689/2002	62	M	728147	OG junction	Ulcerative	Well differentiated Adenocarcinoma
12	521/2002	52	F	710236	OG junction	Fungating	Moderately differentiated infiltrating SCC
13	44/2002	48	F	702940	Lower 1/3rd	Fungating	Well differentiated SCC
14	1879/2002	70	M	732444	Middle 1/3rd	Fungating	Well differentiated SCC
15	1943/2002	47	M	733972	Lower 1/3rd	Fungating	Moderately differentiated infiltrating SCC
16	155/2003	55	F	744644	Lower 1/3rd	Fungating	Well differentiated infiltrating SCC
17	182/2003	50	F	747389	Lower 1/3rd	Fungating	Well differentiated infiltrating SCC
18	397/2003	65	M	746798	Lower 1/3rd	Infiltrative	Poorly differentiated Adenocarcinoma with extensive desmoplasia
19	609/2003	70	M	752181	Lower 1/3rd	Fungating	Moderately differentiated keratinsing squamous cell carcinoma
20	1164/2003	50	M	763403	OG junction	Fungating	Moderately differentiated squamous cell carcinoma
21	1488/2003	60	M	766370	OG junction	Fungating	Moderately differentiated squamous cell carcinoma
22	1539/2003	50	M	769700	Lower 1/3rd	Fungating	Well differentiated SCC
23	1853/2003	55	M	774129	Middle 1/3rd	Fungating	Well differentiated SCC

S. No.	Patho No.	Age	Sex	IP No.	Site of growth	Type of growth	HPE
24	1875/2003	53	M	774012	Lower 1/3rd	Fungating	Well differentiated SCC
25	1988/2003	50	M	776390	Lower 1/3rd	Fungating	Well differentiated SCC
26	2282/2003	45	F	782000	Lower 1/3rd	Fungating	Moderately differentiated SCC
27	721/2004	61	M	795903	Lower 1/3rd	Fungating	Well differentiated Kertanising SCC
28	880/2004	65	F	797632	OG junction	Fungating	Well differentiated Kertanising SCC
29	1012/2004	55	F	797046	Lower 1/3rd	Fungating	Well differentiated Kertanising SCC
30	1324/2004	55	M	801625	Middle 1/3rd	Fungating	Well differentiated Kertanising SCC
31	1437/2004	55	M	804535	OG junction	Fungating	Well differentiated Kertanising SCC
32	1615/2004	48	M	804697	OG junction	Fungating	Well differentiated Kertanising SCC
33	1671/2004	55	M	806105	Upper 1/3rd	Fungating	Well differentiated Kertanising SCC
34	1845/2004	50	M	811003	Lower 1/3rd	Ulcerative	Well differentiated Adenocarcinoma
35	1196/2004	60	M	809567	Middle 1/3rd	Fungating	Well differentiated SCC
36	2181/2004	50	F	815902	Lower 1/3rd	Fungating	Moderately differentiated SCC
37	2131/2004	35	F	814390	Lower 1/3rd	Fungating	Well differentiated SCC
38	2490/2004	60	M	221129	Lower 1/3rd	Fungating	Well differentiated SCC
39	2523/2004	60	M	821477	Lower 1/3rd	Infiltrative	Poorly differentiated SCC
40	2536/2004	65	M	821129	Lower 1/3rd	Infiltrative	Moderately differentiated keratinsing squamous cell carcinoma
41	2638/2004	32	F	820739	Lower 1/3rd	Fungating	Well differentiated SCC
42	2697/2004	60	F	822351	Lower 1/3rd	Fungating	Well differentiated SCC
43	446/2005	50	M	831295	Esophagogastric junction	Fungating	Moderately differentiated SCC
44	825/2005	50	M	838141	Lower 1/3rd	Fungating	Moderately differentiated SCC
45	1503/2005	77	M	845364	Lower 1/3rd	Infiltrative	Well differentiated Adenocarcinoma
46	1790/2005	65	M	849662	Lower 1/3rd	Fungating	Moderately differentiated SCC